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CARDIOLOGY

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VISIT...

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INDEX

HEART FAILURE	2
TTT of HEART FAILURE	14
SYMPTOMS OF CARDIAC DISEASES	26
<i>Dyspnea</i>	28
<i>Cough</i>	32
<i>Hemoptysis</i>	32
<i>Oedema</i>	32
<i>Syncope</i>	34
<i>Chest pain</i>	36
GENERAL EXAMINATION	37
CARDIAC EXAMINATION	41
THE CARDIAC CYCLE	43
VALVULAR HEART DISEASES	45
<i>Mitral stenosis</i>	45
<i>Mitral regurge</i>	53
<i>MVP</i>	55
<i>Aortic stenosis</i>	57
<i>Aortic regurge</i>	61
<i>Tricuspid stenosis</i>	64
<i>Tricuspid regurge</i>	66
CONGENITAL HEART DISEASES	70
DISEASES OF THE PERICARDIUM	85
<i>Dry pericarditis</i>	85
<i>Pericardial effusion</i>	88
<i>Constrictive pericarditis</i>	97
<i>Adhesive pericarditis</i>	100
RHEUMATIC FEVER	101
INFECTIVE ENDOCARDITIS	110
ANATOMY OF THE CORONARIES	120
ISCHEMIC HEART DISEASE	121
<i>Angina Pectoris</i>	125
<i>Acute Myocardial Infarction</i>	138
SYSTEMIC HYPERTENSION	151
PULMONARY HYPERTENSION	162
PULMONARY EMBOLISM	164
AND NOW ARRHYTHMIAS	175
CARDIOMYOPATHIES	227

**“ My doctor is nice; every time I see him,
I'm ashamed of what I think of doctors in
general...”**

~ Mignon McLaughlin

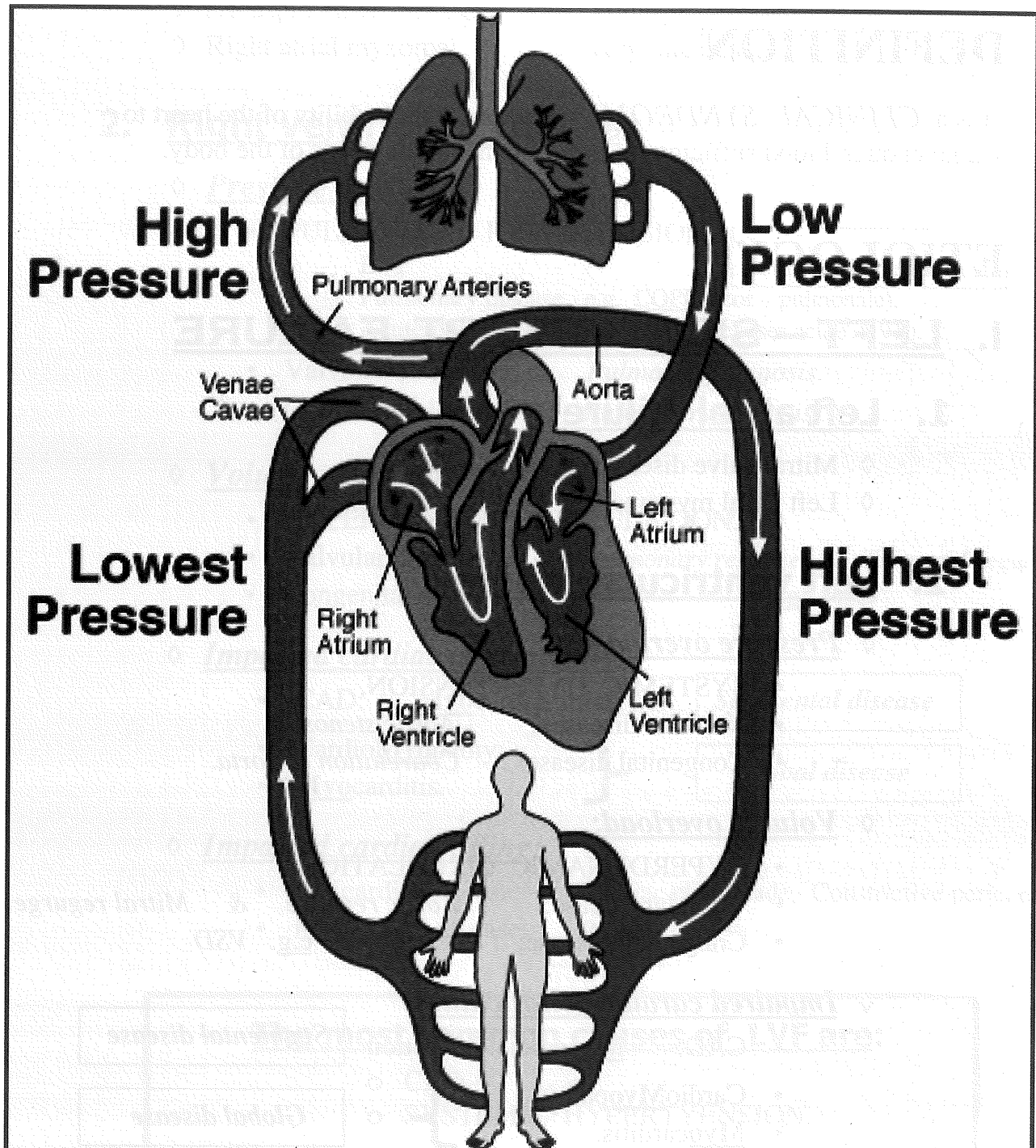
**“ Doctors are men who prescribe medicines
of which they know little, to cure diseases
of which they know less, in human beings
of whom they know nothing...”**

~ Voltaire [Francois-Marie Arouet]

**“ The recommendation for a healthy heart
may one day be: exercise, eat right and
laugh a few times a day...”**

~ Michael Miller, MD

NORMAL CIRCULATION



HEART FAILURE

DEFINITION

It is a CLINICAL SYNDROME resulting from inability of the heart to pump enough blood sufficient to meet the metabolic needs of the body.

ETIOLOGY

I. LEFT – SIDED HEART FAILURE

1. Left atrial failure:

- ◇ Mitral valve disease: MS.
- ◇ Left atrial myxoma: rare cause.

2. Left ventricular failure:

- ◇ Pressure overload:
 - SYSTEMIC HYPERTENSION.
 - Valvular disease: Aortic stenosis.
 - Congenital disease: Coarctation of aorta.
- ◇ Volume overload:
 - HYPERDYNAMIC CIRCULATION.
 - Valvular disease: Aortic regurge & Mitral regurge.
 - Congenital disease: e.g. VSD
- ◇ Impaired cardiac contractility:

- CAD: Myocardial infarction
 - CardioMyopathy.
 - Myocarditis.

}

Segmental disease

Global disease
- ◇ Impaired cardiac filling:
 - Pericardial disease: Cardiac tamponade, Constrictive pericardit
 - Myocardial disease: RCM.

II. RIGHT – SIDED HEART FAILURE

1. Right atrial failure:

- ◇ Tricuspid valve disease: Tricuspid stenosis.
- ◇ Right atrial myxoma: very rare cause.

2. Right ventricular failure:

◇ Pressure overload:

- PULMONARY HYPERTENSION:
 - LVF (the most common cause of RVF).
 - Pulmonary disease, e.g. COPD (cor – pulmonale).
 - Acute pulmonary embolism (causes acute RVF).
- Valvular disease: *Pulmonary stenosis.*
- Congenital disease: *Pulmonary stenosis.*

◇ Volume overload:

- HYPERDYNAMIC CIRCULATION.
- Valvular disease: *Pulmonary regurge & Tricuspid regurge.*
- Congenital disease: e.g. ASD

◇ Impaired cardiac contractility:

- CAD: Myocardial infarction
- CardioMyopathy.
- Myocarditis.

Segmental disease

Global disease

◇ Impaired cardiac filling:

- Pericardial disease: Cardiac tamponade, Constrictive pericarditis.
- Myocardial disease: RCM.

- The most common causes of LVF are:

- CAD.
- SYSTEMIC HYPERTYENSION.

- The most common causes of RVF are:

- LVF.
- PULMONARY DISEASE.

PRECIPITATING FACTORS

- In a patient with compensated HF, these factors may decompensate the heart.
 - During ttt of HF, if these factors are not removed, HF will not improve (**refractory HF**).
-
- ♥ **I**nfections: esp. *infective endocarditis*, *rheumatic activity* & *chest infection*.
 - ♥ **I**atrogenic:
 - **C**orticosteroids & **C**alcium – channel blockers.
 - **D**iscontinuation of anti-failure treatment.
 - **E**xcessive salt intake & IV fluids.
 - ♥ **A**cute myocardial infarction.
 - ♥ **A**rrhythmias.
 - ♥ **A**nemia, thyrotoxicosis & other causes of hyperdynamic circulation.
 - ♥ **P**hysical & emotional stress.
 - ♥ **P**regnancy & late labor.
 - ♥ **P**ulmonary embolism.

PATHOPHYSIOLOGY

1. The clinical manifestations of HF result from:

- ▶ **Forward Failure:** Failure of the heart to eject a sufficient CO:
 - This gives rise to manifestations of **low CO**.
- ▶ **Backward Failure:** Failure of the heart to accept the VR:
 - This gives rise to manifestations of **congestion**.
 - **Pulmonary congestion:** in case of Left – sided HF.
 - **Systemic congestion:** in case of Right – sided HF.

2. Compensatory mechanisms (Cardiac reserve):

- These mechanisms try to restore the cardiac output.
- They are beneficial within limits.
- If they exceed these limits, they will aggravate HF.

► TACHYCARDIA:

1. Mechanism: sympathetic stimulation mediated by:
 - *Marey's law:* *HR is inversely proportional to BP.*
 - *Bainbridge reflex:* *Increased RA pressure causes increased HR.*
2. Effect:
 - *It increases the CO.*
 - *If it is marked ($>160 / \text{min}$):*
it will $\downarrow$ the diastole, $\downarrow$ the ventricular filling & thus $\downarrow$ the CO.

► DILATATION:

- Increased LENGTH of cardiac muscle fibre: in VOLUME overload.
- It increases the force of contraction (*according to Starling's law*).
- If it exceeds certain limits, it will lead to diminished contraction.

► HYPERTROPHY:

- Increased THICKNESS of cardiac muscle fibre: in PRESSURE overload.
- It increases the force of contraction.
- If it exceeds certain limits, the hypertrophied muscle cannot get its adequate blood supply (becomes ischemic) $\rightarrow$ diminished contraction.

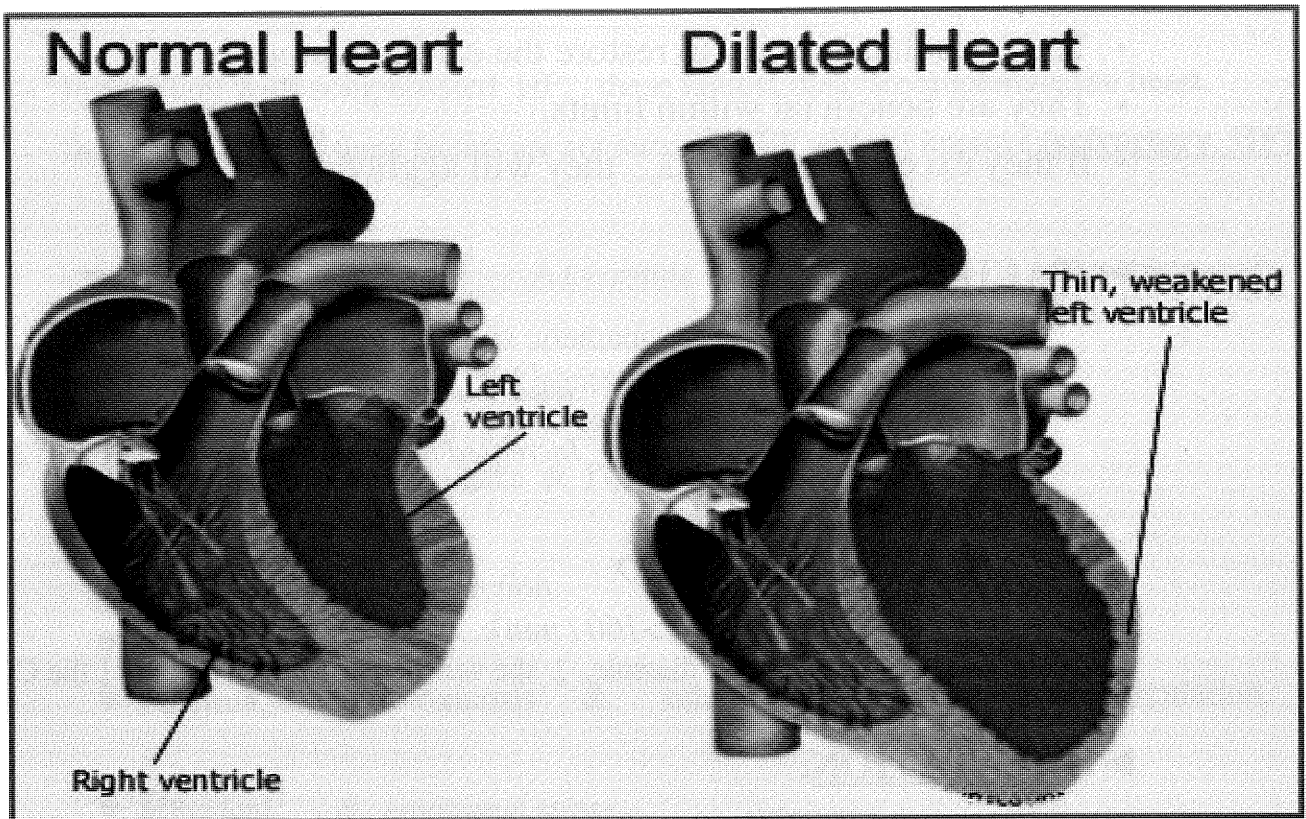
► REDISTRIBUTION OF BLOOD FLOW:

- There is diversion of blood:
 - From less vital organs (e.g. skin & muscles),
 - To more vital organs (e.g. brain & heart).

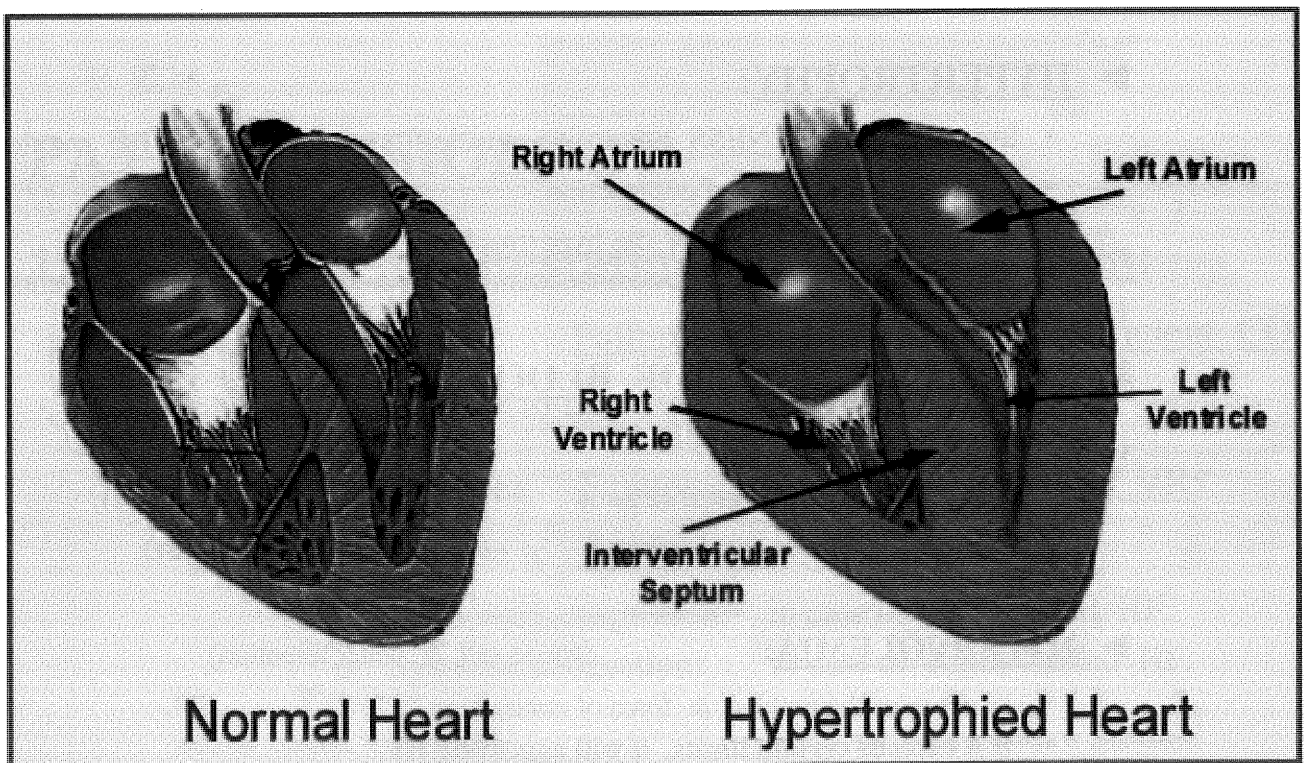
► HYPERVOLEMIA: (due to salt & water retention)

- It increases the preload & force of contraction.
- **If it exceeds certain limits, it will aggravate HF.**

VENTRICULAR DILATATION



VENTRICULAR HYPERTROPHY



3. Neurohormonal changes in HF:



Some of these changes are beneficial, others are harmful:

a) Activation of the **RAAS**

- Beneficial effect: maintains BP & perfusion of vital organs.
- Harmful effects:
 - *The increased angiotensin II: results in vasoconstriction.*
 - *The increased aldosterone: results in salt & water retention.*

b) Activation of the **SNS**

- Beneficial effect: increases the ventricular contractility.
- Harmful effects:
 - *Increases the afterload.*
 - *Induces arrhythmias.*
 - *Causes direct myocardial damage.*

c) The Natriuretic peptide system **(ANP)**

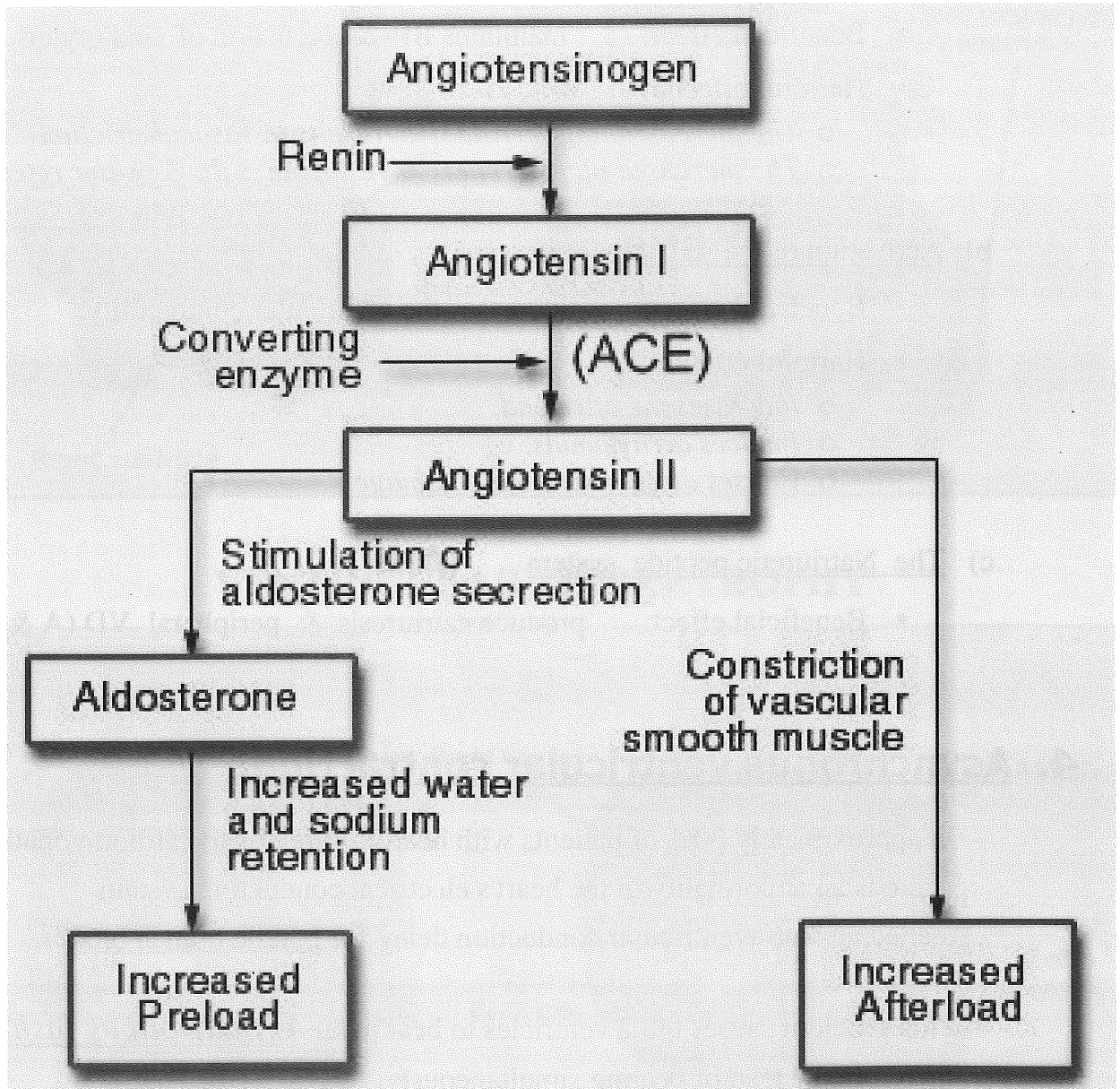
- Beneficial effect: produce natriuresis & peripheral VD (A & V).

4. Asynchronous ventricular contraction:

- In approximately 30% of patients with heart failure due to cardiomyopathy:
There is an abnormality in the heart's electrical conducting system
(called an "intraventricular conduction delay" or bundle branch block).
- This problem causes the 2 ventricles to beat in an Asynchronous fashion:
therefore, instead of beating simultaneously:
 - The 2 ventricles beat slightly out of phase.
 - Also: segmental asynchrony within the LV may occur.

This asynchrony greatly reduces the efficiency of the ventricles in patients with heart failure, whose hearts are already damaged.

RAAS



CLINICAL PICTURE

I. LEFT – SIDED HEART FAILURE

Symptoms

- 1) Ss of low CO: *forward failure.*
- 2) Ss of pulmonary congestion *backward failure.*
- 3) When RVF occurs: *symptoms of pulmonary congestion improve.*

Signs

1) General:

- a) RESPIRATION: Tachypnea (shallow rapid respiration). ★
- b) PULSE:
 - Tachycardia: except in patients receiving digitalis or β-blockers.
 - Small volume: except in conditions of high CO.
 - Pulsus alternans: ★ alternating strong & weak regular beats detected by palpating the pulse or using the sphygmomanometer.
- c) SKIN: Coldness of the skin & Peripheral cyanosis.
- d) CHEST: Pleural effusion & Bilateral basal crepitations. ★

2) Cardiac:

- a) Precordial examination:
 - Signs of LV enlargement.
- b) Auscultation:
 - *Over the mitral area:*
 - Left ventricular gallop (S3): due to flabby myocardium.
 - Pansystolic murmur of functional MR: due to LV dilatation.
 - *Over the pulmonary area:*
 - Signs of pulmonary hypertension.

Manifestations of the cause

e.g. Typical chest pain in Myocardial infarction.

II. RIGHT – SIDED HEART FAILURE

Symptoms

- 1) Symptoms of low CO (forward failure).
- 2) Symptoms of systemic congestion (backward failure).

Signs

1) General:

- a) PULSE:
 - o Tachycardia: except in patients receiving digitalis or β-blockers.
 - o Small volume: except in conditions of high CO.
- b) SKIN: Coldness of the skin & Peripheral cyanosis.
- c) CHEST: Pleural effusion.
- d) PROMINENT SIGNS OF SYSTEMIC CONGESTION:
 - o Neck veins: *Congested pulsating neck veins.*
 - o Liver: *Enlarged, tender, ± pulsating, ± Hepato-jugular reflu.*
 - o Lower Limbs: *Oedema of lower limbs & later on ascites.*
- e) CARDIAC CACHEXIA:

With severe chronic HF, cachexia occurs due to:

 - o ↓ caloric intake: *due to Anorexia, N, V & ↓ absorption (GIT congestion)*
 - o ↑ caloric loss: *due to ↑ metabolic rate (↑ O₂ needs of the failing heart)*

2) Cardiac:

- a) Precordial examination:
 - o Signs of RV enlargement.
- b) Auscultation: (Over the tricuspid area):
 - o Right ventricular gallop (S₃): due to flabby myocardium.
 - o Pancystolic murmur of functional TR: due to RV dilatation.

Manifestations of the cause

e.g. Chronic cough, dyspnea & wheezes in COPD.

CLINICAL CLASSIFICATION

“Clinical categories”

1. LVF versus RVF. (also Biventricular failure).

2. Acute versus chronic HF:

- Most causes produce chronic HF:

Maintained BP
LL oedema
Ventricular enlargement

- Some causes produce acute HF as:

- LVF:
AMI, Acute MR (after AMI or SBE).
- RVF:
Acute pulmonary embolism.
- Biventricular failure:
Acute myocarditis.

↓ BP
No LL oedema
No ventricular enlargement

3. Forward versus backward heart failure.

a) Forward failure:

- There are: manifestations of Low CO.
- Response to ttt: positive inotropics, arteriodilators.

b) Backward failure:

- There are: manifestations of congestion.
- Response to ttt: diuretics, venodilators.

4. Systolic versus diastolic failure:

a) Systolic Failure: ↓ cardiac contractility, e.g. AMI, DCM.

b) Diastolic Failure: ↓ cardiac filling, e.g. CP, RCM, systemic HTN.

5. Low versus high CO failure:

a) Low CO failure:

- There is reduction of CO below normal.
- This type occurs in MOST CASES of HF.

b) High CO failure:

- There is usually peripheral VD & ↓ peripheral resistance, leading to ↑ VR & ultimately ↑ CO.
- There is ↑ CO, however, it is insufficient for the metabolic needs of tissues.
- This type occurs in cases of HYPERDYNAMIC CIRCULATION:
 - o Anemia, AR, Arteriovenous fistula.
 - o Beriberi, PDA, Paget's disease.
 - o Thyrotoxicosis, Liver failure.

6. Etiological classification:

"see before"

FUNCTIONAL CLASSIFICATION

- A classification was developed by the New York Heart Association (NYHA) to assess the SEVERITY of heart failure:

- CLASS I: No limitation of physical activity on ordinary effort.
- CLASS II: Slight limitation of physical activity on ordinary effort.
- CLASS III: Marked limitation of physical activity on less than ordinary effort.
- CLASS IV: Symptoms occur even at rest.

INVESTIGATIONS

I. ECG:

- Chamber enlargement.
- Cause of HF may appear e.g. CAD, BBB.
- Precipitating factor may appear e.g. arrhythmias.

II. IMAGING:

1. CXR:

- Chamber enlargement.
- Pulmonary congestion in left-sided HF, or Pleural effusion.

2. Echocardiography:

- Chamber enlargement.
- Cause of HF may appear e.g. valvular lesions.
- CONTRACTILITY STATE OF THE MYOCARDIUM:
 - o SHORTENING FRACTION (SF)
 - o EJECTION FRACTION (EF)

3. Cardiac catheterization & angiocardiography:

- Chamber enlargement.
- Cause of HF may appear e.g. CAD.

III. Other investigations for the cause & ppt factor:

- Cardiac enzymes: for myocardial infarction.
- CBC: for anemia.
- Thyroid functions: for thyrotoxicosis.

TREATMENT OF HEART FAILURE

GUIDELINES for TTT (PLAN of TTT)

1. Treatment of the underlying etiology..... e.g...
2. Treatment of the precipitating factors..... e.g...

3. Treatment specific to the syndrome of HF:

- Decreasing the cardiac load:

- a) Rest: Physical & emotional.
- b) Reduction of preload:
 - o Diet control, especially salt restriction.
 - o Diuretics.
 - o Vasodilators (Venous).
- c) Reduction of afterload:
 - o Vasodilators (Arterial).

- Decreasing the myocardial damage:

- β -blockers.

- Increasing the myocardial contractility:

- a) Digitalis.
- b) Other inotropic agents:
 - o Sympathomimetic amines: dopamine & dobutamine.
 - o Phosphodiesterase inhibitors: amrinone & milrinone.

- Resynchronizing the ventricles:

“CRT”

- In patients with cardiomyopathy who have the problem of:
"intraventricular conduction delay" or bundle branch block.

- Symptomatic treatment:

- a) For hypoxemia: oxygen administration.
- b) For cardiac dyspnea: aminophylline administration.

4. Treatment of refractory HF.

5. Treatment of acute pulmonary oedema.

PHYSICAL REST

- **BENEFITS:** Bed rest reduces the metabolic needs of the body & the cardiac load.
- **POSITION:** Semisitting position is preferred to decrease the venous return.
- **COMPLICATIONS** of prolonged bed rest:
 - DVT & PULMONARY EMBOLISM.
 - Pneumonia.
 - Constipation & retention of urine.
 - Muscle wasting & osteoporosis.
 - Bed sores.
 - Psychoneurosis.

EMOTIONAL REST

- **SEDATION** may be achieved by: *Diazepam 2 – 5 mg tds orally.*

DIET

- Salt restriction: decreases blood volume & thus decreases the preload.
- Fluid restriction: only needed in severe cases of HF.
- Small frequent meals: to decrease the work of the heart.
- *Reduction of body weight in obese patients.*

VASODILATORS

Action

1. Reduction of preload by: veno – dilatation.
2. Reduction of afterload by: arteriolar dilatation.

Types

VASODILATOR	Site of action	Mode of administration
Nitrates (<i>Isosorbide dinitrate</i>)	Venous	20 – 40 mg / 8h orally
Hydralazine	Arterial	50 mg / 6h orally
Na nitroprusside	Arterial & venous	0.5 – 10 μ g / kg / min, IV infusion
Prazosin (α – blocker)	Arterial & venous	orally
ACE inhibitors: <i>Short acting: Captopril</i> <i>Long acting: Ramipril</i>	Arterial & venous	6.25 – 25 mg / 8h orally 1.25 – 2.5 mg / day orally
ARBs: <i>Candesartan</i>	Arterial & venous	Initially 4, target 32 mg / day orally

Important points

- In chronic HF, ACE – I is the most useful, esp. in **LV systolic failure**.
- **Contraindications to ACE – I in HF:**
 1. Intolerance due to the presence of side effects, e.g. *dry cough*.
 2. HYPERKALEMIA: *serum potassium > than 5.5 mEq/liter.*
 3. Hypotension: *SBP < 90 mm Hg.*
 4. Bilateral renal artery stenosis: *because they will cause renal failure.*
 5. Caution in renal failure with serum creatinine > 3.0 mg/dL & stop the drug if: serum creatinine rises by more than 30 % of the base-line level.
- **When ACE – I is contraindicated in HF,** alternative good VDs are:
 1. Combination of Nitrates (venodilator) & Hydralazine (arterio dilator).
 2. ARBs: *especially Candesartan*, (in case of **intolerance** to ACE – I).

DIURETICS

- They are very useful in the treatment of HF.
- They promote loss of salt & water, thus: ↓ blood volume & ↓ preload.
- **Types:**
 1. Thiazides.
 2. Loop diuretics.
 3. Potassium sparing diuretics.
 4. Others diuretics.

1. Thiazides

- **Action:** on the **distal tubules**, to ↓ reabsorption of: Na, H₂O, K, Cl.
- **Preparations:**
 - Hydrochlorothiazide: 50 – 100 mg/day.
 - Chlorothalidone: 50 – 100 mg/day.
- **Side effects:**
 1. **Hypo** kalemia: which may precipitate digitalis toxicity.
 2. **Hypo** chloremic alkalosis: which may lead to tetany.
 3. **Hypo** natremia.
 4. **Hypo** magnesemia.
 5. **Hyper** uricemia.
 6. **Hyper** glycemia.
 7. **Hyper** lipidemia.
 8. **Hyper** calcemia.

2. Loop diuretics

- **Action:** on the ascending limb of loop of Henle, to ↓ reabsorption of: Na, H₂O, K, Cl.
THEY ARE THE MOST POTENT DIURETICS.
- **Preparations:**
 - Frusemide (Lasix): 40 – 200 mg/day (orally or IV).
 - Ethacrynic acid: 50 – 100 mg/day (orally or IV).
- **Side Effects:**
 - Same as Thiazides (but without hypercalcemia).

3. Potassium sparing diuretics

- **Action:** on the distal tubules to ↓ reabsorption of: Na, & H₂O,
BUT: they have the advantage of retaining K (they ↓ the secretion of K).
THEY ARE WEAK DIURETICS:
 - *Used to potentiate the action of Thiazide or loop diuretics.*
 - *Used to avoid the potassium losing effect of Thiazide or loop diuretics.*
- **Preparations:**
 - Spironolactone (aldosterone antagonist): 100 – 400 mg / day.
 - Triametrine.
 - Ameloridae.
- **Side effects:**
 - Hyperkalemia & Metabolic acidosis.
 - Gynecomastia with prolonged use.

4. Other diuretics

1. Natriuretic peptides:

- Infusions of recombinant NP have been shown to cause:
 - Afferent arteriolar dilation & efferent arteriolar constriction → ↑ GFR.
 - Natriuresis.
 - Peripheral VD (A & V).

2. Carbonic anhydrase inhibitors:

- Diuretics: ↓ reabsorption of: NaHCO₃ in the proximal tubules.
- Also: correct the metabolic alkalosis that occur due to thiazide diuretics.

DIGITALIS

“ Digitalis is no longer extensively used in the management of HF, although it is an effective positive inotropic agent.”

ACTIONS

- ▶ **It increases the myocardial contractility:** *through:*
 Competitive inhibition of sodium-potassium ATPase → ↑ intracellular Na.
 Then, Na-Ca exchange occurs & this increases the intracellular Ca.
 High intracellular Ca promotes sliding of actin & myosin.
 This leads to increased force of myocardial contractility; which causes:
 - Increased cardiac output..
 - Decreased venous congestion.
 - Decreased cardiac size.
- ▶ **It slows the heart rate:** *through:*
 - Vagal stimulation.
 - Direct inhibition of the SAN.
- ▶ **It increases the excitability of the atria & ventricles:**
 - In digitalis toxicity, arrhythmias may occur.
- ▶ **It inhibits the conduction of the AVN:**
 - In digitalis toxicity, AV block may occur.
 - Digitalis can be used to protect the ventricle in atrial arrhythmias.
- ▶ **Diuretic effect:** *through:*
 - Increased renal blood flow.
- ▶ **On ECG:**
 - Digitalis effect: *Sagging depression of ST segment, Flat or inverted T wave.*
 - Digitalis toxicity: *Different types of arrhythmias.*

INDICATIONS

1. Heart Failure.
2. Rapid atrial arrhythmias:
 - *Atrial fibrillation (AF).*
 - *Atrial flutter.*
 - *Supraventricular tachycardia.*

Digitalis is highly indicated in:

- Patients with HF & rapid atrial arrhythmias, esp. AF.
- Patients with HF not responding to: Vasodilators, βB & Diuretics.

CONTRAINDICATIONS

- ABSOLUTE CONTRAINDICATIONS:
 - Digitals toxicity.
 - Ventricular tachycardia (*Digitalis may change it to fatal VF*).
- RELATIVE CONTRAINDICATIONS: “It is better avoided:”
 - Incomplete heart block.
 - Nodal rhythm.
 - Hypertrophic cardiomyopathy.

PREPARATIONS & ADMINISTRATION

1. Oral:

- ▲ It is the usual route of administration:
 - *Digoxin:* excreted mainly by the *kidney* (tab = 0.25 mg).
 - *Digitoxin:* metabolized mainly by the *liver* (tab = 0.1 mg).
- ▲ Dose:
 - Initial dose: 2 tablets daily for 5 days.
 - Maintenance dose: 0.5 – 2 tablets daily.

2. Intravenous:

- ▲ Indicated in:
 - “Acute pulmonary oedema & rapid atrial arrhythmias”.
 - Digoxin (amp. = 0.5 mg).
 - Cedilanid.
 - Ouabain.
- ▲ Dose:
 - 0.5 – 1 mg in 5% glucose over 30 min, THEN:
 - 0.5 mg / 6 h till a therapeutic response appears, THEN:
 - Continue with oral digitalis.

DIGITALIS TOXICITY

1. Clinical Picture:

▲ Gastrointestinal:

- Anorexia: usually the first symptom.
- Nausea & vomiting.

▲ Cardiovascular: “Different types of arrhythmias & heart block”

- **P**remature beats, especially occurring in *bigemini* or *trigemini*.
- **P**aroxysmal atrial tachycardia: *with heart block*.
- **P**aroxysmal ventricular tachycardia: *most serious*.

▲ Neurological:

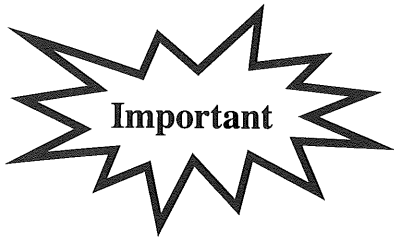
- MENTAL disturbances, e.g. psychosis
- PAIN: headache & neuralgias.
- VISION: **Blurring** of vision & **coloured** vision (yellow or green).

▲ Miscellaneous:

- Increased blood coagulability.
- Gynecomastia, with prolonged use.

2. Treatment:

- Stop digitals.
- Correct hypokalemia, if present:
 - Stop drugs causing hypokalemia. e.g. loop diuretics.
 - Give potassium : Orally or IV.
- Digitalis antibodies.
- TTT of the manifestations of digitalis toxicity:
 - For vomiting: Anti-emetic drugs, e.g. metoclopramide.
 - For arrhythmias:
 1. Anti-arrhythmic Drugs: especially epanutin & lidocaine.
 2. Atropine: for heart block & bradycardia.
 3. DC: Avoided because it may induce more serious arrhythmias.
Allowed ONLY in case of the fatal arrhythmia: VF.



BETA - BLOCKERS



- In the past: they were absolutely contraindicated in HF due to their *-ve inotropic* effect.
- Recently: they are used to treat HF because they were found to:
 - o *Decrease the direct myocardial damage induced by catecholamines in HF.*
 - o *Improve the prognosis.*

- INDICATIONS:

- o **Chronic HF.**
- o **Moderate HF** (NYHA classes II & III).



- CONTRA – INDICATIONS:

- o Acute HF (e.g. shortly after AMI).
- o Severe HF (NYHA class IV).

- ADMINISTRATION:

o TYPES:



Only: **Second generation** β_1 blockers (e.g. Metoprolol), or:
Third generation β_1 blockers (e.g. Carvedilol).

o DOSE: “Gradually increasing doses”



- We start with extremely low doses (one-eighth of the target dose).
- We gradually $\uparrow$ the dose every 1 – 2 weeks to reach the target dose.
- Target dose: 25 – 50 mg of carvedilol daily.

Treatment of Refractory Heart Failure

Etiology of refractory HF:

1. Presence of a mechanical factor hindering the cardiac function: **e.g.**
 - *Severe valvular disease.*
 - *Pericardial effusion or constrictive pericarditis.*
2. Persistence of the cause: **e.g.** Uncontrolled Hypertension.
3. Presence of a precipitating factor: **e.g.** Chest infection.
4. Improper management: **e.g.**
 - *Inadequate rest.*
 - *Inadequate salt restriction.*
 - *Inadequate digitalis.*
5. Terminal cases of HF: with advanced myocardial damage,
e.g. Massive myocardial infarction.

Treatment:

1. Removal of the mechanical factor: **e.g.** surgical ttt of valvular disease.
2. TTT of the cause: **e.g.** proper control of Hypertension.
3. Removal of the precipitating factor: **e.g.** proper antibiotics for chest infection.
4. Proper management:
 - *Adequate rest.*
 - *Adequate salt restriction (< 1gm / day) & fluid restriction in severe cases*
 - *Adequate digitalis.*
5. For Terminal cases of HF:
 - a) *Diuretics:* use combinations e.g. loop diuretic & potassium-sparing diuretic
 - b) *Vasodilators:* use combinations of IV vasodilators such as **Na nitroprusside** and a potent IV inotrope such as **dopamine** or **dobutamine**
 - c) *Mechanical measures:*
 - Mechanical removal of fluid using phlebotomy or dialysis.
 - Mechanical assistance of circulation using intra-aortic balloon counterpulsation
“ Balloon is inflated in diastole & deflated in systole ”.
 - d) *Cardiac transplantation:* in patients below the age of 60.

Treatment of Acute Pulmonary Oedema

1. Hospitalization in ICU: bed rest in sitting position.
 2. Oxygen administration: 6 – 8 litres / min .
 3. **Morphine IV** 5 mg, to relieve the anxiety and decrease the sympathetic stimulation, thus causing VD.
 4. **Frusemide IV** 40 mg, to correct the hypervolemia, to be repeated every ½ hour until relief.
- The 2 most important measures*
5. Vasodilators **IV**:
 - *Na nitroprusside*: **IV infusion** (0.5 – 10 µg / kg / min).
 - *Nitroglycerine*: **IV infusion.**
 6. Positive inotropics **IV**:
 - *Digitals*, *esp. in cases associated with AF.*
 - *Dopamine or Dobutamine.*
 7. Aminophylline **IV** (250 – 500 mg), slowly, to relieve the bronchospasm.
 8. Treatment of the cause & the precipitating factor.
 9. In refractory conditions:
 - *Mechanical removal of fluid using phlebotomy or dialysis.*
 - *Mechanical assistance of circulation using intra-aortic balloon counterpulsation.*
 - *Mechanical ventilation.*

Resynchronizing the ventricles:

“CRT”

Indication

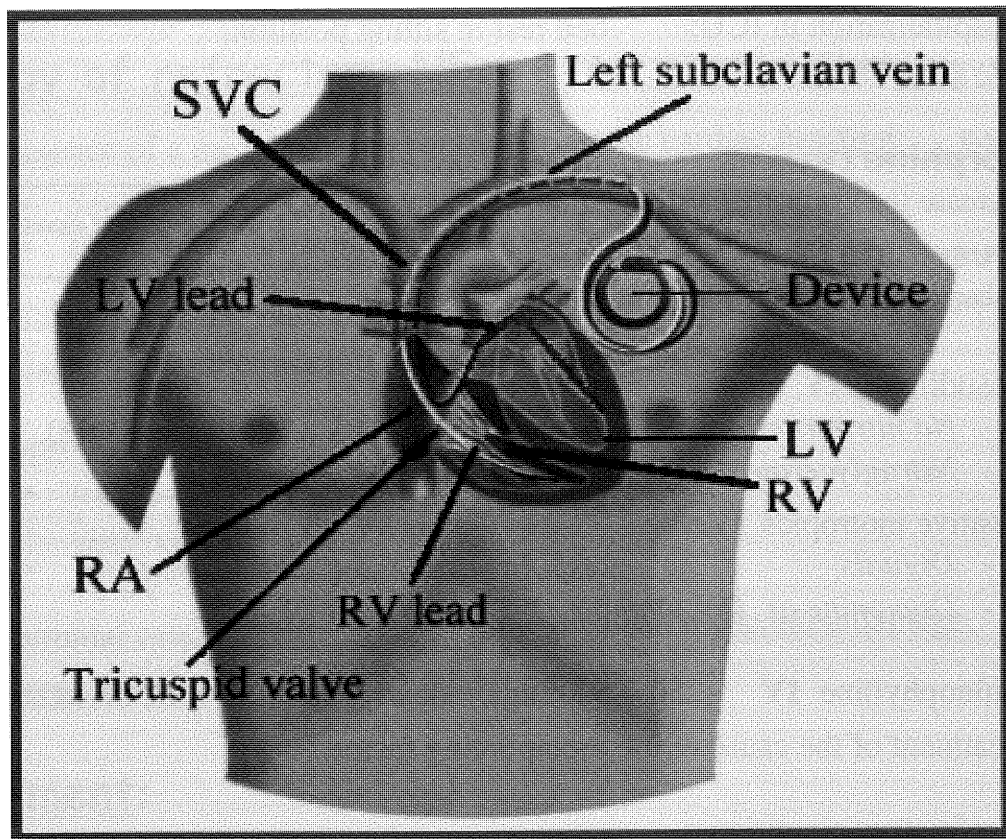
- In patients with cardiomyopathy who have the problem of:
"intraventricular conduction delay" or bundle branch block:
 - As evidenced by wide QRS ≥ 0.12 sec in ECG.
 - In patients with severe symptoms (NYHA class III, IV) despite proper ttt.

Method

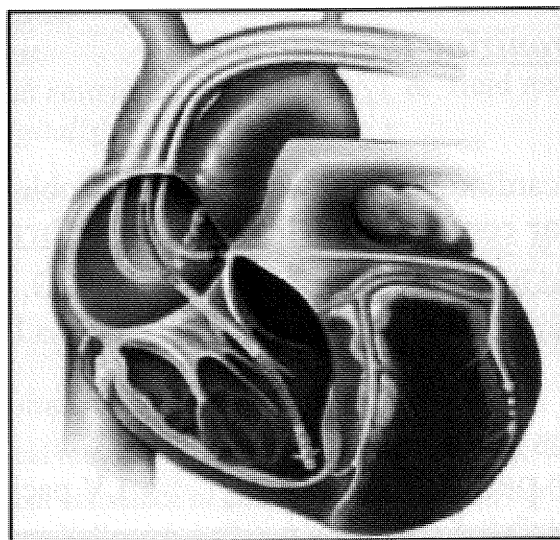
- Biventricular pacing or - LV pacing

CRT = Cardiac Resynchronization Therapy

CARDIAC RESYNCHRONIZATION THERAPY “CRT”



Coated wires called leads carry electrical energy from the CRT device to the heart. This energy helps the RV and LV to pump at the same time (Synchronized).



Important points in Heart failure

- ♥ CAD is the most common cause of LVF.
- ♥ LVF is the most common cause of RVF.
- ♥ Symptoms of LVF improve when RVF develops.
- ♥ Pulsus alternans is an important sign of LVF.
- ♥ SF is an important echocardiographic indicator of the cardiac function & is measured as follows:

$$SF = \frac{LVDD - LVSD}{LVDD}$$

- LVDD is the LV diastolic diameter
- LVSD is the LV systolic diameter

- ♥ The 3 main lines of treatment are :
 - To **decrease:** the preload.
 - To **decrease:** the afterload.
 - To **decrease:** the myocardial damage.
 - To **INCREASE:** the myocardial contractility.
- ♥ β -blockers are recently considered:
 - An effective measure in the treatment of HF.

Clinical manifestations of Cardiac Disorders

Symptoms of cardiac disorders

1. PULMONARY CONGESTION SYMPTOMS:

- These occur in *left sided heart failure* due to failure of the heart to accept the *pulmonary* venous return:
 - Dyspnea.
 - Cough.
 - Hemoptysis.

2. SYSTEMIC CONGESTION SYMPTOMS:

- These occur in *right sided heart failure* due to failure of the heart to accept the *systemic* venous return:
 - Confusion & insomnia.
 - Right hypochondrial pain.
 - Anorexia, nausea, vomiting, dyspepsia & epigastric pain.
 - Oedema of dependent parts (LLs or sacrum).
 - Abdominal swelling due to ascites.

3. LOW CARDIAC OUTPUT SYMPTOMS:

- These occur in *left or right sided heart failure or in both* due to failure of the heart to eject a sufficient CO:
 - BRAIN: Dizziness, headache & syncope.
 - EYES: Blurring of vision.
 - HEART: Angina pectoris.
 - KIDNEYS: Oliguria.
 - MUSCLES: Easy fatiguability & Intermittent claudication.
 - SKIN: Pallor & coldness; peripheral cyanosis in severe cases

4. PALPITATION:

- This is an unpleasant awareness of the heartbeat.
- This occurs due to:
 - Change in rate: e.g. *Tachycardia or Bradycardia.*
 - Change in rhythm: *Arrhythmia.*
 - Change in force: *Forceful cardiac contraction.*
 - Psychoneurosis.

5. CHEST PAIN:

- Pain of cardiac origin may arise from:
 - Myocardium: e.g. angina pectoris or AMI.
 - Pericardium: e.g. pericarditis or pericardial effusion.
 - Endocardium: e.g. mitral valve prolapse.
 - Aorta: e.g. dissecting aortic aneurysm.
 - Pulmonary: e.g. acute pulmonary embolism.

6. PRESSURE SYMPTOMS:

- These occur due to pressure of enlarged cardiac chambers on the surrounding structures.
- An enlarged left atrium, aortic aneurysm or pericardial effusion may compress:
 - Oesophagus: dysphagia.
 - Tracheobronchial tree: dyspnea & cough.
 - Left RLN: hoarseness of voice.
 - Spine: back pain.
 - SVC: swelling of the face & puffiness of eye lids.

7. CYANOSIS:

- Bluish discolouration of skin & may be mucous membranes due to the presence of more than 5 gm reduced (unoxygenated) Hb / 100 cc of blood.

DYSPNEA

- It is a subjective distress associated with difficulty in breathing.

TYPES

1. Exertional: 5 grades (American thoracic society scale)

- Grade 0: none no dyspnea.
- Grade 1: mild dyspnea on **m**arked exertion.
- Grade 2: moderate dyspnea on **m**oderate exertion.
- Grade 3: severe dyspnea on **m**ild exertion.
- Grade 4: very severe dyspnea on **m**inimal exertion.

2. Dyspnea at rest.

3. Orthopnea:

- **What happens ??**
 - o It is dyspnea on recumbency that is relieved by sitting up.
- **Pathogenesis**: recumbency results in:
 - o ↑ VR to the heart → more pulmonary congestion & more dyspnea.
 - o Elevation of the diaphragm & improper use of chest wall muscles.

4. Paroxysmal nocturnal dyspnea: PND

- **What happens ??**

Classic PND:

- The patient is aroused 1 – 2 hours after sleeping with:
 - o Severe dyspnea, Sweating, Cyanosis, Cough,
 - o Expectoration: frothy blood-tinged.
- The patient sits up in bed or stands beside a window to get more air.

Cardiac asthma:

- Generalized wheezes: due to bronchial oedema & bronchospasm.

Acute Pulmonary oedema:

- Generalized bubbling crepitations: due to intra-alveolar oedema.

- **Pathogenesis:**
 - o Recumbancy increases the VR to the heart → more pulmonary congestion.
 - o Decreased sympathetic activity during sleep → reduction of inotropism.
- **Incidence:**
 - PND occurs more commonly in acute LVF as it is an acute process.
 - PND occurs less commonly in MS because it is a **gradual process** allowing time for **protective mechanisms** to take place to ↓ further pulmonary congestion.
 - a. **Reflex vasoconstriction of pulmonary arteries:**

This will decrease the pulmonary congestion, but on the other hand it will lead to development of pulmonary hypertension.
 - b. **Anastomosis between pulmonary & bronchial veins:**

This will cause shunting of high pressure & congestion from the pulmonary veins to the bronchial veins.
This may lead to formation of BRONCHIAL VARICES which may rupture on coughing or straining causing severe hemoptysis.
 - c. **Development of interstitial pulmonary barrier:**

Chronic pulmonary congestion → to haemosiderosis & fibrosis.
This will decrease transudation of fluid from pulmonary capillaries.
 - d. **Thickening of the walls of pulmonary vessels.**

Differentiation between Cardiac & Bronchial asthma

	CARDIAC	BRONCHIAL
<i>Age</i>	Any age	Usually young age
<i>Past history</i>	Cardiac symptoms	Chest symptoms
<i>Duration</i>	Short	Long
<i>Time of the attack</i>	1 – 2 hours after sleep	Early in the morning
<i>Sputum</i>	Frothy, may be blood – tinged	Thick pellets
<i>Chest examination</i>	Generalized wheezes, Bilateral basal crepitations	Generalized wheezes
<i>Heart examination</i>	Gallop & murmurs	Normal
<i>ECG</i>	Abnormal	Normal
<i>DRUGS</i>		
<i>Adrenaline</i>	<i>Contraindicated</i>	<i>Improves the condition</i>
<i>Morphine</i>	<i>Improves the condition</i>	<i>Contraindicated</i>
<i>Aminophylline</i>	<i>Improves the condition</i>	<i>Improves the condition</i>

ACUTE PULMONARY OEDEMA

DEFINITION

- Rapid accumulation of fluid in the interstitial & intra-alveolar spaces of the lung.

ETIOLOGY

1. CARDIOGENIC PULMONARY OEDEMA:

Sudden increase in the *pulmonary venous pressure*:

- Acute left sided heart failure (LSHF): e.g. AMI.
- Acute exacerbation of chronic LSHF: e.g. MS with a ppt. factor as AF.

2. NON - CARDIOGENIC PULMONARY OEDEMA: (ARDS)

Sudden increase in the *pulmonary capillary permeability*:

- **SEPTICEMIA**, Pneumonia (severe), Pancreatitis.
- **STROKES**, Head injuries.
- **SHOCK**.
- **TRAUMA**, Burns.
- **TERMINAL** Liver & renal failure.
- INHALATION of gases (e.g. chlorine), Aspiration of gastric contents.
- DIC.

3. OTHER CAUSES:

- Sudden expansion of a collapsed lung, e.g. *rapid aspiration of massive pleural effusion*.
- Severe hypoalbuminemia.

CLINICAL PICTURE

1. Manifestations of the cause: e.g.

- o Typical chest pain in AMI (Cardiogenic pulmonary oedema).
- o Neurological manifestations in STROKE (Non - Cardiogenic pulmonary oedema).

2. Typical Manifestations of Acute Pulmonary Oedema:

- o Severe dyspnea at rest & orthopnea.
- o Sweating & marked irritability.
- o Central cyanosis.
- o Cough with expectoration of excessive frothy blood-tinged sputum.
- o Generalized BUBBLING CREPITATIONS.
- o Generalized wheezes.

INVESTIGATIONS

1. Investigations of the cause: **e.g.**

- o ECG & Cardiac enzymes for AMI (*Cardiogenic pulmonary oedema*).
- o Brain imaging (CT scan) for STROKE (*Non - Cardiogenic pulmonary oedema*).

2. CHEST X – Ray:

- o Exaggerated pulmonary vascular markings especially in upper lung zones.
- o Haziness of lung fields.
- o Kerley B lines.

3. ARTERIAL BLOOD GASES (ABG):

- o PO₂: Decreased.
- o PCO₂: Decreased at first, *then*, Increased lately in severe cases.

TREATMENT

1. Treatment of the cause: **e.g.**

- o Reperfusion (revascularization) for AMI (*Cardiogenic pulmonary oedema*).
- o General care, Antiplatelets for STROKE (*Non - Cardiogenic pulmonary oedema*).

2. Treatment of Cardiogenic pulmonary oedema:

- o Refer to the section of HEART FAILURE.

3. Treatment of Non - Cardiogenic pulmonary oedema:

- o Mechanical ventilation.
- o IV Corticosteroids.

COUGH

- In the cardiac patient, cough is usually due to:
 - Pulmonary congestion.
 - Pulmonary infection.
 - Pulmonary infarction.

HEMOPTYSIS

- In the cardiac patient, hemoptysis is usually due to:
 - Pulmonary congestion.
 - Pulmonary infection.
 - Pulmonary infarction.
 - Rupture of bronchial varices.
 - Rupture of aortic aneurysm.

OEDEMA

- Pathogenesis of cardiac oedema:

1. Increased capillary hydrostatic pressure:

A) Increased venous pressure: due to stagnation of blood in systemic veins.

B) Salt & water retention due to:

1. Decreased GFR: due to ↓ renal blood flow, due to ↓ CO.

2. Increased Aldosterone: due to:

- ↑ *renin release due to:* reduced renal blood flow.
- ↑ *stimulation of:* volume receptors of Aldosterone.
- ↓ *hepatic catabolism of:* Aldosterone.

3. Increased ADH: due to:

- ↑ *stimulation of:* volume receptors of ADH.
- ↓ *hepatic catabolism of:* ADH.

2. Increased capillary permeability: due to hypoxia of the capillary walls.

3. Decreased plasma oncotic pressure due to:

- o *Decreased protein intake due to:* anorexia & vomiting.
- o *Decreased protein absorption due to:* intestinal congestion.
- o *Decreased protein formation due to:* liver congestion.

- **Characteristics of cardiac oedema:**

1. **Occurs in the dependant parts of the body:**
 - o Ankle oedema: in ambulant patients.
 - o Sacral oedema: in bed ridden patients.
2. **Bilateral: but one side may be affected more due to:**
 - o Deep venous thrombosis.
 - o Postural, in patients sleeping on one side.
3. **Pitting.**
4. **Associated with other features of cardiac disease.**
5. **Oedema of lower limbs always precedes appearance of ascites:**
except in two conditions in which ascites occurs first "**Ascites precox**":
 - a) **Pericardial effusion & constrictive pericarditis:**
 - Kinking of hepatic veins causes early liver congestion & ascites.
 - Obstruction of lymphatics passing through the central tendon of the diaphragm causes accumulation of lymph in the peritoneum.
 - b) **Tricuspid regurge:**
 - Regurgitation of blood causes liver congestion & ascites.

- **Differential diagnosis of cardiac oedema:**

1. **Renal Oedema:**

It occurs in GN, e.g. *Nephrotic* or *Nephritic syndrome*.

Oedema occurs first in eye lids & is associated with features of renal disease.

2. **Hepatic Oedema:**

Oedema of LLs occurs after ascites & is associated with features of liver disease.

3. **Nutritional Oedema:**

It occurs in severe nutritional deficiency & is associated with other features of nutritional deficiencies.

4. **Angioneurotic Oedema:**

- This **allergic** oedema occurs with constant relation to certain factors, e.g. *eating certain food*, or *inhaling certain substances*.
- **Acute onset** especially in: **L**ips, **L**ids, **L**arynx, but may be generalized.
- There is usually +ve family history of: oedema or other forms of allergy.
- The patient himself may have: other forms of allergy.
- There is characteristic rapid response to: **anti-allergic measures**.

SYNCOPE

DEFINITION

Loss of consciousness “SUDDEN & TRANSIENT” due to acute cerebral hypoxia.
There is inability to maintain postural tone & is followed by spontaneous recovery.

ETIOLOGY

1. Vasomotor syncope

A) Vasovagal syncope (Neurocardiogenic syncope):

- It results from severe vagal stimulation which leads to:
Severe bradycardia, hypotension, pallor & sweating.
- It results from: Sudden severe fear, pain, trauma (e.g. to testicles).
- It is the most common cause of syncope & is known as simple fainting

B) Carotid sinus syndrome:

- It results from pressure on hypersensitive carotid sinus baroreceptors:
e.g. *During shaving.*

2. Cardiac syncope

(any cause of low CO) especially:

- Aortic stenosis (or any other valvular obstruction)
- Acute heart failure (e.g. AMI).
- Arrhythmias (whether tachy- or brady – arrhythmias).
- Adams-stokes attacks.

3. Cerebral syncope

(reduced cerebral blood flow)

- Vertebrobasilar TIAs.

4. Hypoxic syncope

(↓ O₂ content of the cerebral blood flow)

- Fallot's tetralogy & other cyanotic diseases or severe anemia.

5. Postural syncope

(Orthostatic syncope)

- Normally, reflex VC of blood vessels of LLs occurs on standing to prevent pooling of blood in LLs.
- This effect is mediated through sympathetic stimulation.
- If this mechanism is defective, BP will be markedly lowered in the standing position (postural hypotension) & syncope may occur.
- Causes include:
 - Autonomic neuropathy, e.g. DIABETES.
 - DRUGS: sympatholytic drugs (e.g. Ganglion blockers), VD.
 - Lumbar sympathectomy.
 - Hyponatremia.
 - Prolonged recumbency.
 - Elderly patients.

6. Situational syncope

- Rare syncope caused by a variety of activities in susceptible individuals:
 - *Cough syncope* (*Tussive syncope*).
 - *Micturition syncope* (*more common in old men especially at night*).
 - *Defecation syncope*.
- Underlying mechanism:

Straining → decreased VR → decreased CO → Syncope.

DIFFERENTIAL DIAGNOSIS

1. Anxiety attacks: (panic attacks with feeling of faintness or dizziness):
 - o They are not relieved by recumbency.
 - o They are associated with: *anxiety, palpitation & hyperventilation.*
2. Hypoglycemic attacks:
 - o History: of DM with overdose of insulin or missed meal.
 - o Features: of sympathetic overactivity as sweating & palpitation.
3. Epileptic attacks (Seizures): refer to Neurology.

CHEST PAIN

CARDIOVASCULAR CAUSES OF CHEST PAIN:

- **Myocardium:**
CAD: *angina or AMI.*
- **Pericardium:**
Pericarditis or pericardial effusion.
- **Endocardium:**
Mitral valve prolapse.
- **Aorta:**
Dissecting aortic aneurysm.
- **Pulmonary:**
Acute pulmonary embolism.
- **Pain of cardiac neurosis:**
 - Type of patient: *neurotic patients especially young ♀.*
 - Site & Reference: *Left inframamary, Localized.*
 - Character & Duration: *Stitching, of variable duration.*
 - Precipitating factors: *No relation to exertion.*
 - Relieving factors: *Not relieved by rest.*
 - Associations:
 - o *Features of neurosis: especially sighing respiration.*
 - o *Normal cardiac examination.*
- **Huge cardiomegaly:**
Rarely causes retrosternal heaviness.

Signs of cardiac disorders

I. GENERAL EXAMINATION

The general examination of the cardiac patient includes looking for the following signs:

1. **Decubitus**

- | | |
|--|---------------------------|
| 1. Orthopnea: | left-sided heart failure. |
| 2. Prayer's position (sitting up and leaning forward): | pericardial effusion. |
| 3. Squatting: | the Tetralogy of Fallot. |

2. **Pallor**

In cardiac disease, pallor may be due to:

1. Anemia.
2. Rheumatic activity.
3. Infective endocarditis.
4. Low cardiac output.
5. Shock.

3. **Cyanosis**

1. **Central cyanosis**, occurs in:
 - o Congenital heart disease.
 - o Hypoxic cor pulmonale.
2. **Peripheral cyanosis**, occurs in:
 - o Venous stagnation, e.g. *Right sided HF*.
 - o Venous obstruction.

4. **Jaundice**

In cardiac disease, jaundice may be due to:

1. Severe right sided heart failure.
2. Pulmonary embolism.

5. **Fever**

In cardiac disease, fever may be due to:

1. Rheumatic activity.
2. Infective endocarditis.
3. Pericarditis.
4. Myocarditis.
5. Myocardial infarction.
6. Pulmonary infarction.
7. Pulmonary infection.

6. **Clubbing of digits**

In cardiac disease, it occurs in:

1. Infective endocarditis (pale clubbing).
2. Cyanotic cardiac conditions (cyanotic clubbing).

7. **NECK VEINS**

They should be examined for:

1. Pressure: (Congested or not ??)

- It should not exceed 3 cm vertically above the level of the sternal angle.
- Normally, the neck veins are not congested.

2. Pulsations: (Pulsating or not ??)

- Normally, the neck veins are pulsating with systolic collapse.
- Normally, the pulsations are wavy & consist of:
 - a-wave: right atrial contraction.
 - x-descent: right atrial relaxation.
 - c-wave: transmitted from adjacent carotids.
upward bulge of tricuspid valve into right atrium.
 - v-wave: RA filling.
 - y-descent: RA emptying.

CLINICAL SIGNIFICANCE OF NECK VEINS:

1. Normally:

- They are non congested, & are pulsating with systolic collapse.

2. Abnormally:

1) Abnormal pressure: “ Congested neck veins ”

❖ Pulsating:

- o Right sided HF.
- o Pericardial effusion & constrictive pericarditis.
- o Hypervolemia.

❖ Non-pulsating:

- o SVC obstruction (SVC thrombosis or Mediastinal syndrome).

2) Abnormal Pulsations: “ Abnormal waves ”

❖ a- wave:

- o Absent : AF.
- o Giant: Pulmonary hypertension, PS, TS.
- o Cannon waves:
 - *Regular:* *Nodal rhythm.*
 - *Occasional:* *A-V dissociation.*

❖ x-descent:

- *Obliterated* (systolic expansion of neck veins):
 - * TR.
 - * AF.
- *Prominent:*
 - * Constrictive pericarditis.
 - * Pericardial effusion.

❖ y-descent:

- *Prominent:*
 - * Constrictive pericarditis.

8. **Oedema of lower limbs**

- o Right sided HF.
- o Pericardial effusion & constrictive pericarditis.

9. **Abdomen**

1. Hepatomegaly:

- Enlarged tender: Right sided HF & pericardial disease.
- Enlarged tender & pulsating: TR & TS.
- Enlarged tender & sharp: Cardiac cirrhosis.

2. Splenomegaly:

- Infective endocarditis (tender).
- Right sided HF & pericardial disease.

3. Ascites.

II. Cardiac Examination

A) AIM OF CARDIAC EXAMINATION:

To detect:

1. Evidence of generalized cardiac enlargement:

- Shift of the apex outwards lateral to the MCL.

2. Evidence of right ventricular enlargement:

- a) Apex beat: (outermost lowermost impulse)
 - Diffuse
 - Shifted outwards
 - Shows systolic retraction : accompanied by reciprocal left parasternal uplift
(**right ventricular rocking**)
- b) Left parasternal & epigastric pulsations.
- c) Dullness over the lower half of the sternum.

3. Evidence of left ventricular enlargement:

- a) Apex beat:
 - Localized
 - Shifted outwards & downwards
 - Shows systolic bulge: accompanied by reciprocal left parasternal retraction
(**left ventricular rocking**)
- b) Apex impulse:
 - Heaving (forcible , sustained): pressure overload.
 - Hyperdynamic (forcible , non-sustained): volume overload.
 - Hypodynamic (weak): myocardial disease

4. Evidence of great vessel dilatation:

- a) Dilated pulmonary artery:
 - Dullness & pulsations in the second left space.
- b) Dilated aorta:
 - Dullness & pulsations in the second right space.

5. Evidence of endocardial affection (valvular affection):

- Presence of specific murmurs & thrills.

6. Evidence of myocardial affection:

- Presence of S₃ gallop over the mitral or tricuspid areas in LV or RV affection respectively.

7. Evidence of pericardial affection:

- Refer to the chapter of “ Pericardial disease ”

8. Evidence of pulmonary hypertension:a) Precordial examination:

- Pulsations , diastolic shock & dullness in the second left space.

b) Auscultation:

- S₂ is accentuated.
- Systolic ejection click.
- Systolic ejection murmur.
- Soft early diastolic murmur due to functional pulmonary regurge (Graham Steel).
- S₄ over the tricuspid area.

9. Evidence of arrhythmias:

- Change in the rate or rhythm of the heart beats.

B) TECHNIQUE OF CARDIAC EXAMINATION:

“ Refer to the Clinical notes ”.

THE CARDIAC CYCLE

- **First Heart Sound:**

The cardiac cycle starts by contraction of the ventricles resulting in the closure of the mitral & tricuspid valves producing the *first heart sound* (S_1).

- **Isometric Contraction Phase:**

The ventricles continue to contract while the 4 valves of the heart are closed, so the pressure inside the ventricles rises rapidly without change in the volume.

- **Ejection Phase:**

- When the pressure in the ventricles exceeds the pressure in the aorta & pulmonary artery, the aortic & pulmonary valves will open (*normally with no sound*).
- Then the blood rushes from the ventricles to the aorta and pulmonary artery, first rapidly "maximum ejection phase", then slowly "reduced ejection phase".

- **Second Heart Sound:**

After evacuation of blood, the ventricles relax, so the pressure in the aorta and pulmonary artery will exceed the pressure in the ventricles resulting in closure of aortic & pulmonary valves producing the *second heart sound* (S_2).

- **Isometric Relaxation Phase:**

The ventricles continue to relax while the 4 valves of the heart are closed, so pressure inside the ventricles falls rapidly without change in the volume.

- **Ventricular Filling Phase:**

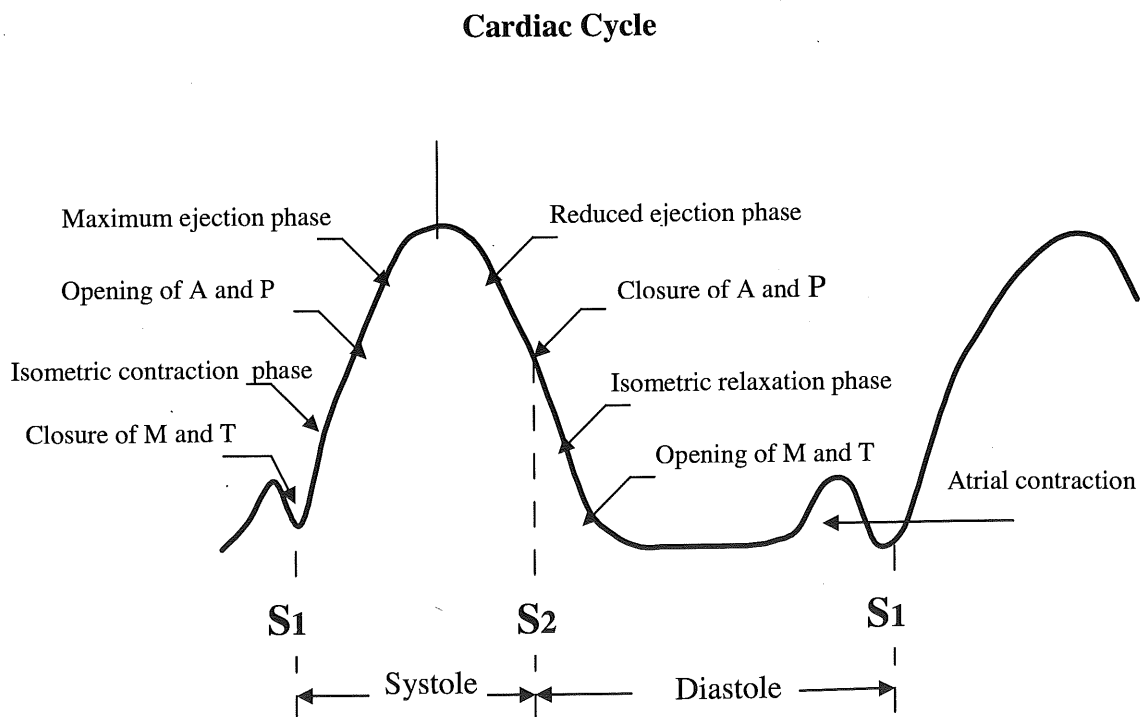
When the pressure in the ventricles becomes lower than the pressure in the atria, blood flows to the ventricles passively, at first rapidly: *“maximum filling phase”*, then slowly: *“reduced filling phase”*.

- **Atrial Systole:**

The last amount of blood in the atria is pushed actively by atrial contraction in the late diastole *“presystole”*.

- **Ventricular contraction:**

Occurs again & the cycle is repeated.



VALVULAR HEART DISEASES

MITRAL STENOSIS

ETIOLOGY

1. Rheumatic Fever: the most common cause.
2. Congenital: a rare cause.
3. Relative stenosis: (not an organic stenosis)
 - *Increased blood flow through the mitral valve.*
 - *Carey-Coombs murmur in acute rheumatic valvulitis.*
 - *Austin flint murmur in severe aortic regurge.*

PATHOPHYSIOLOGY

- In mild cases:
The blood flow through the mitral valve remains normal.
No symptoms occur.
- In severe cases: (valve area less than 2cm^2):
The blood flow through the mitral valve is decreased.
Blood stagnates in pulmonary veins (**pulmonary congestion**).
- Later on:
Vasoconstriction of pulmonary arterioles occurs to ↓ pulmonary congestion,
but this will lead to: **pulmonary hypertension**.
- Finally:
RV enlargement & then RVF will occur secondary to pulmonary HTN.

Therefore four stages will occur in patients with MS:

STAGES OF MITRAL STENOSIS

- **Stage I:** “ Mild or asymptomatic mitral stenosis ”
The only abnormality is anatomical narrowing of the mitral valve,
but the patient is fully compensated (*no symptoms*).
- **Stage II:** “ Mitral stenosis with pulmonary congestion ”
The *pulmonary venous pressure* is elevated.
- **Stage III:** “ Mitral stenosis with pulmonary hypertension ”
The *pulmonary arterial pressure* is elevated.
- **Stage IV:** “ Mitral stenosis with right ventricular failure ”

CLINICAL PICTURE

There is a ***latent period*** of several years between the initial attack of rheumatic carditis & the development of manifestations of mitral stenosis.

Symptoms

Does the patient with MS always symptomatize ???

1. **Stage I:**
No symptoms.
2. **Stage II:**
Symptoms of pulmonary congestion (*but pulmonary oedema is not common*).
Symptoms of low CO.
3. **Stage III:**
Symptoms of pulmonary congestion: improve.
Symptoms of low CO: increase.
4. **Stage IV:**
Symptoms of systemic congestion.

Dyspnea is the most common symptom

Signs

General:

1. Stage I: no signs.
2. Stage II: signs of: pulmonary congestion.
3. Stage III: signs of: low cardiac output, *malar flush*, *giant a-wave*.
4. Stage IV: signs of: systemic congestion.

Cardiac:

A] Precordial examination:

Stage I & Stage II:

- Apex: normal site & slapping character.
- Diastolic thrill: ending in a palpable S₁.

Stage III & IV:

- The previous findings.
- Signs of: pulmonary hypertension.
- Signs of: right ventricular enlargement.

B] Auscultation:

► Stage I & Stage II (Over the mitral area):

1. Accentuated first heart sound: due to:

- Fibrosis of the mitral cusps.
- Forcible closure of the mitral cusps: because:
 - o *They are displaced downwards due to high LA pressure.*



Diminished S₁ in mitral stenosis denotes:

- o Double mitral lesion (with predominant regurge) or
- o Calcified mitral valve.

2. Mitral opening snap:

- It is **sharp & snapping** due to opening of the rigid cusps of mitral valve.
- It is heard in the **early diastole**:
 - o *Just after S₂ (separated from it by the isometric relaxation phase).*
 - o *Just before the murmur of mitral stenosis.*
- Its presence denotes **non-calcificaton** of the mitral valve.

3. Murmur of mitral stenosis:

- *Timing:*
 - Mid-diastolic presystolic with presystolic accentuation.
 - In AF, there is loss of presystolic accentuation (due to loss of atrial contraction).
- *Character:* rumbling.
- *Site:* at or slightly inside the apex.
- *Propagation:* not propagated.
- *Position:*
 - Best heard with the cone of the stethoscope.
 - In the left lateral position.

4. Silent Mitral Stenosis (MS with no murmur) due to:

- a. High LV pressure: **e.g.** associated LVF.
- b. Low LA pressure: **e.g.**
 - *Severe pulmonary hypertension.*
 - *RVF.*
- c. In association with ASD.

► Stage III

- The previous findings.
- Auscultatory findings of pulmonary hypertension:
 - a) Over the pulmonary area:
 - S₂ is accentuated.
 - Systolic ejection click.
 - Systolic ejection murmur.
 - Soft early diastolic murmur: *due to functional PR.*
(Graham Steel murmur)
 - b) Over the tricuspid area:
 - S₄.

► Stage IV

- The previous findings.
- Auscultation over tricuspid area:
 - *Pan systolic murmur of functional tricuspid regurge.*
 - *RV gallop (S₃) due to RVF.*

COMPLICATIONS

1. In the mitral valve:

- Rheumatic activity.
- Calcification.
- Infective endocarditis.

2. In the left atrium:

- a) *Arrhythmias*, especially AF.
- b) *LA enlargement*, causing pressure symptoms:
 - on oesophagus : dysphagia.
 - on left bronchus : dyspnea & cough.
 - on left recurrent laryngeal nerve: hoarseness of voice.
- c) *Thrombo-embolic complications*:
 - Systemic embolization: e.g. cerebral, peripheral, renal.
 - Ball & valve embolus: leading to syncope & sudden death.

3. In the right ventricle:

- RVF.

4. In the left ventricle:

- No LVF in isolated mitral stenosis.

5. In the lung:

- Hemoptysis.
- Pulmonary infection.
- Pulmonary embolism (secondary to DVT).
- Pulmonary oedema is not common in mitral stenosis (see before).

6. Complications of surgery.

INVESTIGATIONS

1. CXR:

- a) Stage I: no abnormality.
- b) Stage II: (PA view & lateral view with Barium)
 - o LA enlargement.
 - o Pulmonary congestion.
- c) Stage III & IV:
 - o Right ventricular enlargement & RA enlargement.
 - o Pulmonary hypertension.
 - o May be calcified mitral valve.

2. ECG:

- a) Stage I : No abnormality.
- b) Stage II: LA enlargement (P mitrale: broad & bifid).
- c) Stage III & IV:
 - *Right atrial enlargement* (P pulmonale: tall & peaked).
 - *Right ventricular enlargement.*

3. Echocardiography:

- The most sensitive & specific non-invasive method diagnosing MS.
- Detects the severity of stenosis by measuring:
 - o *The valve area.*
 - o *The pressure gradient across the valve.*
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiography:

- Detects the severity of stenosis by measuring:
 - o *The valve area.*
 - o *The pressure gradient across the valve.*
- Detects chamber enlargement.

WHEN DO WE SAY TIGHT MITRAL STENOSIS??

▼ Tight MS is diagnosed:

- According to the stage:
 - Stage II with dyspnea more than grade II, or
 - Stage III, or
 - Stage IV.
- According to the echocardiography:
 - Valve area less than 1 cm^2 .

▼ Tight MS is an indication for SURGERY .

TREATMENT

1. Medical:

- a) Prophylaxis against: Rheumatic activity, Infective endocarditis (uncommon).
- b) Symptomatic for: Complications e.g. HF, AF, infection, embolization.

2. Surgical:

- a) Indications:
 1. Tight mitral stenosis (valve area is $< 1 \text{ cm}^2$).
 2. Marked symptoms not responding to adequate medical treatment.
 3. Embolization with no serious deterioration of the condition of the patient.
- b) Types of operations:
 1. Mitral commissurotomy: closed or open.
 2. Valve replacement:
 - By a prosthesis (tissue or synthetic).
 - Indications:
 - Calcification .
 - Associated mitral regurge.
 - Recurrent stenosis after commissurotomy.

c) Complications:

1. Embolization.
2. Arrhythmias.
3. Mitral incompetence.
4. Restenosis.
5. Post – cardiectomy syndrome:
 - Pleuro-pericarditis that may occur 10 – 15 days following the operation.
 - It is possibly an autoimmune response to damaged cardiac tissue during surgery.
6. Complications of artificial valves:
 - Infective endocarditis.
 - Thrombo-embolism.
 - Mechanical dysfunction.
 - Hemolytic anemia.

3. **Balloon dilatation:**

- May be performed in some patients indicated for commissurotomy.

- **Important question:** **“Self – assessment”**
 “What are the changes induced by AF in a case of MS” ??

MITRAL REGURGE

ETIOLOGY

A] Organic:

1. Rheumatic fever: *the most common cause.*
2. Congenital.
3. Prolapse of the mitral valve (MVP).
4. Papillary muscle dysfunction e.g. CAD.
5. Infective endocarditis.
6. Iatrogenic: *following mitral commissurotomy.*

B] Relative:

- Dilatation of the mitral ring secondary to LV dilatation.

PATHOPHYSIOLOGY

1. During systole:

A part of blood regurgitates from LV to LA leading to:

- Low CO.
- LA dilation: due to increased blood volume in LA.

2. During diastole:

A large volume of blood → LV → LV enlargement which may end in LVF.

CLINICAL PICTURE

Symptoms

1. No symptoms: *in early cases.*
2. Symptoms of low cardiac output: *in late cases.*
3. Symptoms of pulmonary congestion: *in late cases.*
4. PALPITATION.

Palpitation is the most common symptom

Signs

General:

- No signs: *in early cases.*
- Signs of low cardiac output: *in late cases.*
- Signs of pulmonary congestion: *in late cases.*

Cardiac:

A] Precordial examination:

- Signs of LV enlargement: *with hyperdynamic apex.*
- Systolic thrill: *over the apex.*

B] Auscultation:

1. First heart sound: **weak** (muffled) *due to failure of proper mitral closure.*
2. Third heart sound: **present** *due to excessive flow of blood from LA to LV.*
3. Murmur of mitral regurge:
 - *Timing:* pan systolic starting with S₁.
 - *Character:* soft or harsh.
 - *Site:* over the apex.
 - *Propagation:*
 - To the axilla.
 - To the base of the heart & medially in posterior leaflet disease.
 - *Position:* heard best in the left lateral position.

COMPLICATIONS

Same complications of MS,

but:

1. Infective endocarditis: *is common.*
2. Left ventricular failure: *occurs.*

INVESTIGATIONS

1. CXR:

- No abnormality: *in early cases.*
- Enlarged LA & LV: *in late cases.*
- Pulmonary congestion: *in late cases.*
- Calcified mitral valve especially: *in late cases.*

2. **ECG:**

- Enlarged LA (P mitrale: broad & bifid).
- Enlarged LV.

3. **Echocardiography:**

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: e.g. MVP.

4. **Cardiac catheterization & angiocardiography:**

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: e.g. CAD.

TREATMENT

1. **Medical:**

- Same as that of mitral stenosis.

2. **Surgical:**

- Valve replacement.

Mitral Valve Prolapse

(Barlow's syndrome – Floppy mitral valve)

- It is more common in young females.
- Mild prolapse is regarded as a normal variant.

ETIOLOGY

- **Unknown**, but there is familial incidence.
- Isolated abnormality, but may be associated with: Marfan's syndrome or ASD.

PATHOPHYSIOLOGY

During ventricular systole, a mitral valve leaflet (commonly the posterior) prolapses into the LA. This may result in papillary muscle strain & some mitral regurge.

Usually the case is not hemodynamically significant.

CLINICAL PICTURE

Symptoms

- ASYMPTOMATIC.
- Atypical chest pain: *is the most common symptom.*
- Palpitation.

Signs

- Mid systolic click.
- Late systolic murmur.

COMPLICATIONS

- Progressive mitral regurgitation.
- Infective endocarditis.
- Arrhythmias.
- Systemic embolization.
- Sudden death.

INVESTIGATIONS

- Echocardiography is diagnostic.

TREATMENT

- Surgery: mitral valve replacement in cases with severe MR
- Prophylaxis against: infective endocarditis.
- Anti – arrhythmic therapy.
- Propranolol: is effective for chest pain.
- Reassurance.

AORTIC STENOSIS

ETIOLOGY

1. Rheumatic fever.
2. Calcific.
3. Congenital:
 - It may be: valvular, subvalvular or supravalvular.
4. Hypertrophic cardiomyopathy (Idiopathic Hypertrophic Subaortic Stenosis; IHSS):
 - It produces a subvalvular obstruction in the systole due to: contraction of the hypertrophied interventricular septum.
5. Relative stenosis (not an organic stenosis):
 - a) Dilatation of the aorta:
Hypertension, atherosclerosis, aortic aneurysm.
 - b) Increased blood flow across the aortic valve:
Hyperdynamic circulation: e.g. AR.

PATHOPHYSIOLOGY

- During systole, there is obstruction of blood flow from LV to aorta leading to:
 1. Low cardiac output.
 2. Pressure overload on LV leading to LVH & LVF.
- Normally, the aortic valve area is 3-4 cm². In severe AS, it is less than 0.8 cm².

CLINICAL PICTURE

Symptoms

1. No symptoms: *in mild cases.*
2. Symptoms of low CO: *in severe cases.*
3. Symptoms of pulmonary congestion: *due to LVF.*
4. SYNCOPE: especially exertional.
5. ANGINA: due to:
 - *Reduced coronary blood flow: due to low CO & shortened diastole.*
 - *Left ventricular hypertrophy: increases the myocardial O₂ demands.*
 - *Associated coronary atherosclerosis: especially in calcific AS.*

Signs

General:

1. Pulse:
 - Pulsus parvus et tardus (plateau pulse): *rises slowly, of small volume, returns slowly.*
 - Pulsus bisferiens: *bifid pulse occurring in double aortic lesion.*
2. Systolic thrill: over the carotid arteries.
3. Signs of low CO: in severe cases (esp. low SBP).
4. Signs of pulmonary congestion: due to LVF.

Cardiac:

A] Precordial examination:

1. Signs of LVH: with a heaving apex.
2. Systolic thrill: over second right space → apex & carotid arteries.

B] Auscultation:

- Over the aortic area:
 1. Second heart sound: weak.
 2. Systolic ejection click: due to opening of the rigid cusps.
 3. Systolic ejection murmur:
 - Midsystolic, harsh.
 - Maximum over second right space → apex & carotid arteries.
- Over the pulmonary area:
 - Reversed splitting of the second heart sound.
- Over the mitral area:
 1. S₄.
 2. Propagated murmur of AS.

COMPLICATIONS

1. LVF.
2. Infective endocarditis.
3. Sudden death: usually due to VF.
4. Heart block: in calcific AS due to extension of calcification to the AV bundle.
5. Rheumatic activity: in rheumatic AS.

INVESTIGATIONS

1. **CXR:**

- No abnormality: *in mild cases.*
- LV: LVH.
- Lungs: Pulmonary congestion when LVF occurs.
- AORTA:
 - *Small aortic knuckle **or** post-stenotic dilatation.*
 - *Aortic valve calcification may be seen.*

2. **ECG:**

- LVH.

3. **Echocardiography:**

- Detects the severity of stenosis by:
 - *Measurement of valve area.*
 - *Measurement of pressure gradient across the valve.*
- Detects the type of stenosis.
- Detects LVH.

4. **Cardiac catheterization & angiocardiography:**

- Detects the severity of stenosis by:
 - *Measurement of valve area.*
 - *Measurement of pressure gradient across the valve.*
- Detects the type of stenosis.
- Detects LVH.

	Congenital	Rheumatic	Calcific
Age	Young	Young & Middle	Old
History of RF	Absent	Present	Absent
Type	Valvular or Subvalvular or Supravalvular	Valvular	Valvular
Associations	Congenital anomalies	Other valvular lesions	Atherosclerosis

TREATMENT

1. **Medical:**

- Same as that of mitral stenosis.
- Anginal attacks: may be relieved by SL nitrates.

2. **Surgical:** (Aortic valve replacement) *Indications:*

1. Presence of severe symptoms.
2. Pressure gradient more than 50 mmHg.
3. Valve area less than 0.8 cm^2 .

3. **Balloon dilatation:** *Indications:*

1. Children: with congenital AS as an alternative to surgery.
2. Elderly: with severe calcific AS who are too ill to undergo surgery.

AORTIC REGURGE

ETIOLOGY

1. Rheumatic fever: *the most common cause.*
2. Infective endocarditis.
3. Congenital.
4. Dilatation of the ascending aorta as in:
 - a) Syphilitic aortitis: *syphilis produces dilatation of the aortic ring.*
 - b) Severe hypertension.
 - c) Ankylosing spondylitis.
 - d) Aortic aneurysm: *with dissection.*
 - e) Marfan syndrome.

PATHOPHYSIOLOGY

- Regurgitation of blood from the aorta to the LV in diastole leads to:
 - 1) Volume overload on the LV leading to LV dilatation & later on LVF.
 - 2) ↑ SBP: due to ↑ LV stroke volume.
 - 3) ↓ DBP.
- ↑ SBP & ↓ DBP → wide pulse pressure → peripheral signs of AR.

CLINICAL PICTURE

Symptoms

1. Generalized BODY THROBBING: due to increased arterial pulsations.
2. PALPITATION: due to forcible LV contraction.
3. Symptoms of pulmonary congestion: when LVF occurs.
4. Angina pectoris : “Two types of angina occur in aortic regurge”
 - Classic angina of effort:
Decreased DBP: reduces coronary filling.
 - Angina of Lewis:
Nocturnal & associated with autonomic disturbances e.g. sweating & tachycardia.

Signs

General:

“Peripheral signs of aortic regurge”

1. De-Musset sign: nodding of the head.
2. Corrigan's sign: prominent carotid pulsations.
3. Systolic thrill: over the carotid arteries.
4. Water hammer pulse: *rises rapidly, of big volume, collapses rapidly.*
5. Capillary pulsations: detected in nail bed, lips or ear lobule.
6. Pistol shots: loud booming sounds heard with each pulse beat over the arteries (especially femoral) due to sudden distension of collapsed arteries.
7. Duroziez's sign:
 - ♣ It consists of systolic & diastolic murmurs over the femoral artery if it is slightly compressed with the stethoscope.
 - ♣ The systolic murmur is due to the rapid flow of blood to the periphery, while the diastolic murmur is due to rapid regurge of blood to the heart.
8. Blood Pressure:
 - a) Wide pulse pressure:
 - Exaggerated difference between systolic & diastolic blood pressure.
 - b) Hill's sign:
 - Exaggerated difference between SBP in LLs & ULs: more than 50 mmHg.
 - Normally, SBP in LLs is higher than in ULs: by about 10 – 20 mmHg

Cardiac:

A] Precordial examination:

- Signs of LV enlargement: with hyperdynamic apex.
- No thrill over the aortic area: in isolated AR.

B] Auscultation:

- Over the aortic area:
 1. Normal second heart sound.
 2. MURMUR of AR:
 - *Timing*: early diastolic, decrescendo.
 - *Character*: soft blowing.
 - *Site*: maximum over the third space.
 - *Propagation*: to the apex.
 - *Position*: best heard with the diaphragm of the stethoscope, the patient is:
 - Sitting up.
 - Leaning forward.
 - Holding his breath in forced expiration.
 3. Soft ejection systolic murmur:
 - Due to ↑ blood flow across the aortic valve (relative AS).

- Over the Mitral area:

1. S₃.
2. Propagated murmur: of AR.
3. Pan systolic murmur: of functional MR.
4. Austin Flint murmur: (mid-diastolic) due to:

♦ *Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative MS.*

COMPLICATIONS

1. Rheumatic activity.
2. Infective endocarditis.
3. LVF.

INVESTIGATIONS

1. CXR:

- LV enlargement & dilated aorta “**Aortic configuration** (**Boot-shaped heart**)”.
- Lungs: Pulmonary congestion when LVF occurs.

2. ECG:

- LV enlargement.

3. Echocardiography:

- Detects the severity of the lesion.
- Detects LV enlargement.

4. Cardiac catheterization & angiocardiography:

- Detects the severity of the lesion.
- Detects LV enlargement.

TREATMENT

1. Medical:

- Same as that of mitral stenosis.
- Syphilis: anti – syphilitic ttt.

2. Surgical: (Aortic valve replacement) Indications:

1. Presence of severe symptoms.
2. Progressive cardiomegaly.
3. Declining LV functions.

TRICUSPID STENOSIS

ETIOLOGY

- Rheumatic fever: the most common cause, & is almost always associated with MS.
- Carcinoid syndrome: a rare cause.

PATHOPHYSIOLOGY

- During diastole, there is obstruction of blood flow from RA to RV leading to:
 - Systemic venous congestion.
 - Low CO.

CLINICAL PICTURE

Symptoms

1. Systemic venous congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - Congested pulsating neck veins with giant A wave.
 - Enlarged tender pulsating liver.
 - Ascites *before* oedema of lower limbs (ascites precox).
2. Low CO.

Cardiac:

A] Precordial examination:

- RA enlargement (dullness to the right border of the sternum).
- RV enlargement (due to frequently associated MS).

B] Auscultation:

Over tricuspid area:

- Accentuated first heart sound.
- Opening snap.
- Mid-diastolic murmur that increases by inspiration.

INVESTIGATIONS

1. **CXR:**

- RA & RV enlargement.

2. **ECG:**

- RA & RV enlargement.

3. **Echocardiography:**

- Diagnostic.

4. **Cardiac catheterization & angiocardigraphy:**

- Diagnostic.

TREATMENT

1. **Medical:**

- TTT of right sided – HF.

2. **Surgical:**

- Valve replacement.

TRICUSPID REGURGE

ETIOLOGY

1. Functional: **the most common cause** due to dilatation of the tricuspid ring secondary to RV dilatation.
2. Organic: **rare:**
 - Rheumatic fever.
 - Infective endocarditis.
 - Congenital.
 - Carcinoid syndrome.

PATHOPHYSIOLOGY

- During systole, blood regurgitates from RV to RA causing:
 - Low CO .
 - RA enlargement.
 - RV enlargement.
 - RVF (systemic congestion).

CLINICAL PICTURE

Symptoms

1. Systemic congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - *Congested pulsating neck veins:* *with systolic expansion.*
 - *Enlarged tender pulsating liver.*
 - *Ascites **before** oedema of LLs* *(ascites precox).*
 - *Mild jaundice & peripheral cyanosis* *(cyano – icteric face).*
2. Low CO.

Cardiac:

A] Precordial examination:

- RA & RV enlargement .
- Rarely: systolic thrill over tricuspid area.

B] Auscultation: “ Over tricuspid area ”

1. Weak muffled S₁.
2. S₃.
3. Pan systolic murmur:
 - *Timing:* pan systolic.
 - *Character:* soft or harsh.
 - *Site:* tricuspid area.
 - *Propagation:* to the apex, but not to the axilla.
 - *Caravallo's sign:* *murmur increases with inspiration being of right sided origin.*

INVESTIGATIONS

1. CXR:

- RA & RV enlargement.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiography:

- Diagnostic.

TREATMENT

1. Medical:

- TTT of right sided – HF.

2. Surgical:

- Valve replacement.

PULMONARY STENOSIS

A] Organic:

- Congenital: the most common cause.
- Carcinoid syndrome: rare.

B] Functional:

- Pulmonary hypertension.

PULMONARY REGURGE

A] Functional:

- Mostly due to PH which causes dilatation of the pulmonary ring.

B] Organic:

- Congenital.
- Carcinoid syndrome.

Valve Lesion	Most important cause	Most important symptom	Most important sign	CXR	ECG
MS	Rheumatic	Pulm. Congestion esp. <u>Dyspnea</u>	- Accentuated S₁ - Mid-diastolic rumbling <u>M</u> over apex - Pulmonary hypertension	LAE Pulm. congestion	P-mitrale P-pulmonale AF
MR	Rheumatic	<u>Palpitation</u>	- Pan systolic <u>M</u> & thrill over apex propagated to axilla	LAE LVE	LAE LVE
AS	Rheumatic Calcific Congenital	Low CO esp. <u>Angina</u> & <u>Syncope</u>	- Heaving apex - Harsh ejection systolic <u>M</u> & thrill “over second right space → carotid”	LVE Small aortic knuckle or Post-stenotic dilatation	LVE
AR	Rheumatic Syphilitic	Throbbing Palpitation	- Peripheral signs - Hyperdynamic apex - Early diastolic soft blowing <u>M</u> “over the third left space”	Boot-shaped heart	LVE
TS	Rheumatic	Systemic congestion	- Giant a-wave - Mid-diastolic rumbling <u>M</u> over tricuspid “↑ with inspiration”	RAE	RAE
TR	Functional	Systemic congestion	- Systolic expansion in neck veins - Pansystolic <u>M</u> over tricuspid “↑ with inspiration”	RAE RVE	RAE RVE

CONGENITAL HEART DISEASES

CRITERIA TO SUSPECT CONGENITAL HEART DISEASES:

- 1. **A**bsence of history of rheumatic fever.
- 2. **B**asal or parasternal thrills or murmurs.
- 3. **C**ardiac manifestations before the age of 5 years.
- 4. **C**entral cyanosis or systemic hypertension in infancy or early childhood.
- 5. **D**extrocardia without mediastinal shift.

THESE DISEASES MAY BE CLASSIFIED ACCORDING TO:

- 1. The presence or absence of **cyanosis**.
- 2. The **hypertrophied (overloaded) ventricle**.

CLASSIFICATION

The hypertrophied ventricle	Non-cyanotic	Cyanotic
1. Right ventricular hypertrophy	a) Pulmonary stenosis b) Atrial septal defect	a) Triology of Fallot b) Eisenmenger's syndrome
2. Left ventricular hypertrophy	a) Aortic stenosis b) Coarctation of the aorta c) Patent ductus arteriosus	a) Tricuspid atresia
3. Biventricular hypertrophy	a) Venticular septal defect	a) Transposition of the great arteries b) Truncus arteriosus
4. Absence of ventricular hypertrophy	a) Any mild lesion b) Dextrocardia	a) Tetralogy of Fallot

PULMONARY STENOSIS

TYPES

1. Valvular:

- The most common type.
- Commonly there is post-stenotic dilatation.

2. Subvalvular: infundibular.

3. Supravalvular: the rarest.

PATHOPHYSIOLOGY

- Resistance to flow of blood from RV to pulmonary artery causes:
 - ❖ Low CO.
 - ❖ RV hypertrophy then RVF.

CLINICAL PICTURE

Symptoms

1. Low CO.
2. RVF.

Signs

General:

1. Signs of low CO.
2. Signs of low RVF.
3. Giant a-wave.

Cardiac:

A] Precordial examination:

- Signs of RV hypertrophy.
- Systolic thrill over the pulmonary area.
- Dullness without pulsations over the pulmonary area in valvular PS (due to post-stenotic dilatation).

B] Auscultation:

- Over the pulmonary area:
 - Weak S₂ with wide splitting.
 - Ejection systolic click (in valvular PS).
 - Ejection systolic murmur.
- Over the tricuspid area:
 - S₄.

COMPLICATIONS

1. Infective endocarditis.
2. RVF.
3. Pulmonary TB due to lung oligemia.

INVESTIGATIONS**1. CXR & screen:**

- RA & RV enlargement.
- Dilated non pulsating pulmonary artery (post-stenotic dilatation)
- Lung oligemia.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardigraphy:

- Diagnostic.

TREATMENT**Medical:**

- Prophylaxis against infective endocarditis.
- Treatment of RVF.

Surgical: (In severe cases: pressure gradient more than 50 mmHg)

- Valvular stenosis: Valvotomy.
- Infundibular stenosis: Infundibular resection.

Balloon Valvotomy: may be done in: Valvular stenosis.

ATRIAL SEPTAL DEFECT

- A communication between the 2 atria.

PATHOPHYSIOLOGY

- LA pressure is higher than RA pressure, so part of blood is shunted from LA to RA which also receives blood coming from the venae cavae, leading to RA dilatation (volume overload).
- This volume overload will then pass to the RV (RV dilatation), then to the pulmonary artery (pulmonary artery dilatation), then to the lung (lung plethora).
- Later on, vasoconstrictive & obliterative changes occur in the pulmonary arterioles to decrease the lung plethora, but this will lead to pulmonary hypertension.
- Therefore RA pressure will increase till it exceeds the LA pressure causing reversal of the shunt & central cyanosis (Eisenmenger's syndrome).

CLINICAL PICTURE

Symptoms

1. Absent in small defects.
2. Dyspnea & recurrent chest infections (due to lung plethora).
3. Symptoms of RVF in late cases.
4. Symptoms of low CO in severe cases.

Signs

General:

1. Giant a-wave due to pulmonary hypertension.
2. Systemic congestion signs due to RVF.

Cardiac:

A] Precordial examination:

- Pulsations & dullness over pulmonary area.
- Signs of RV enlargement.

B] Auscultation:

- Over the pulmonary area:

1. Accentuated S₂ (pulmonary hypertension).
2. **Wide fixed splitting of S₂:**
 - ❖ **Wide** splitting **is due** to increased RV blood volume which will delay the evacuation of the RV, delay closure of the pulmonary valve and delay P₂.
 - ❖ **Fixed** splitting is due to the inability of inspiration to further increase the RV blood volume, **because** the inspiratory increase in venous return is compensated by an equivalent decrease of the left-to-right shunt, thus keeping the pulmonary blood flow constant.
3. Systolic ejection murmur (due to increased pulmonary blood flow).

- Over the tricuspid area:

1. S₃ (due to increased RV blood flow).
2. Middiastolic murmur (due to increased blood flow across tricuspid).

COMPLICATIONS

1. Recurrent chest infections.
2. RVF.
3. AF.
4. Eisenmenger's syndrome : *shunt reversal.*
5. Lutembacher's syndrome : *congenital ASD & acquired MS.*

INVESTIGATIONS

1. CXR & screen:

- Dilatation of RA & RV & pulmonary artery.
- Lung plethora.
- **Hilar dance:** marked pulsations of the pulmonary artery & its branches under the screen.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Detects the defect.
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiography:

- Increased pressure in right side of the heart.
- Increased O₂ tension in RA than in the venae cavae.
- The catheter may pass from RA to LA through the ASD.
- Angiocardiography will show the defect.

TREATMENT

Medical:

- Prophylaxis against infective endocarditis.
- Treatment of complications.

Surgical:

- Closure of the defect.

VENTRICULAR SEPTAL DEFECT

1 SMALL VSD (ROGER'S DISEASE)

- ♥ It is a small defect in the muscular part of the interventricular septum.
- ♥ It is of no clinical significance, except that it may cause infective endocarditis.
- ♥ It is diagnosed by a loud pan systolic murmur & thrill over the left 3rd & 4th spaces.

2 BIG VSD

- ♥ This occurs in the membranous part of the interventricular septum.

PATHOPHYSIOLOGY

- LV pressure is higher than RV pressure, so part of blood is shunted from LV to RV which also receives blood coming from RA, leading to RV enlargement (volume overload).
- This volume overload will then pass to the pulmonary artery (pulmonary artery dilatation) then to the lung (lung plethora).
- Later on, vasoconstrictive & obliterative changes occur in the pulmonary arterioles to decrease the lung plethora, but this will lead to pulmonary hypertension.
- Therefore RV pressure will increase till it exceeds LV pressure causing reversal of the shunt & central cyanosis (Eisenmenger's syndrome).

CLINICAL PICTURE

Symptoms

1. Dyspnea & recurrent chest infections (due to lung plethora).
2. Biventricular failure in late cases.
3. Low CO in severe cases.
4. Palpitation.

Signs

General:

1. Giant a-wave (pulmonary hypertension).
2. Signs of biventricular failure.

Cardiac:

A] Precordial examination:

- o Biventricular enlargement with hyperdynamic apex.
- o Systolic thrill over 3rd & 4th left spaces.
- o Pulsations & dullness over the pulmonary area.

B] Auscultation:

- Over the 3rd & 4th spaces:
 - Harsh pan systolic murmur.
- Over the pulmonary area:
 - Accentuated S₂.
 - Widely split S₂.
 - Systolic ejection murmur.
- Over the mitral area:
 - S₃.
 - Mid diastolic murmur (due to ↑ blood flow across mitral valve).

COMPLICATIONS

1. Recurrent chest infections.
2. RV & LV failure.
3. Infective endocarditis.
4. Eisenmenger's syndrome.

INVESTIGATIONS

1. CXR & screen:

- Biventricular enlargement.

2. ECG:

- Biventricular enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiography:

- Increased pressure in RV & pulmonary artery.
- Oxygen tension is higher in RV than in RA.
- The catheter may pass through the VSD.
- Angiocardiography demonstrates the defect.

PATENT DUCTUS ARTERIOSUS

- Persistence of fetal communication between the left pulmonary artery & the aorta, just distal to the left subclavian artery.

PATHOPHYSIOLOGY

- Since the aortic pressure is higher than the pulmonary pressure in both systole & diastole, blood will be shunted from aorta to pulmonary artery in both systole & diastole, leading to:
 - Increased pulmonary flow causing pulmonary artery dilatation.
 - Increased LA flow causing LA enlargement.
 - Increased LV filling causing LV enlargement.
 - Increased LV SV causing increased systolic BP.
 - Diastolic shunt of blood from aorta to pulmonary causing decreased diastolic BP.
 - Later on, pulmonary hypertension & reversal of the shunt occur.

CLINICAL PICTURE

Symptoms

1. Dyspnea & recurrent chest infections (lung plethora).
2. Symptoms of LVF in late cases.

Signs

General:

1. Big pulse volume & water-hammer pulse.
2. Giant a-wave due to pulmonary hypertension.
3. Differential cyanosis (cyanosis only in lower limbs with shunt reversal).

Cardiac:

A] Precordial examination:

- LV enlargement with hyperdynamic apex.
- Continuous thrill over the left infraclavicular area.
- Pulsations & dullness over the pulmonary area.

B] Auscultation:

- Over the left infraclavicular area:
 - Gibson's murmur: continuous machinery murmur propagated to the pulmonary area.

- Over the pulmonary area:
 - Accentuated S₂.
 - Reversed splitting of S₂.
- Over the mitral area:
 - S₃.
 - Mid diastolic murmur.

COMPLICATIONS

1. LVF.
2. Infective endocarditis .
3. Eisenmenger's syndrome.

INVESTIGATIONS

1. CXR & screen:

- LA & LV enlargement.
- Dilated pulmonary artery.
- Lung plethora.

2. ECG:

- LA & LV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiography:

- Diagnostic.

TREATMENT

Medical:

- Prophylaxis for infective endocarditis.
- Treatment of complications.

Surgical:

- Closure of the ductus.

COARCTATION OF THE AORTA

- Congenital narrowing in a part of the descending aorta .

CLINICAL SIGNIFICANCE

- It is an important cause of secondary hypertension.

COMPLICATIONS

1. Secondary hypertension.
- 2.. LVF.
3. Infective endocarditis .
4. Dissection or rupture of aorta.

DIAGNOSIS

- Harsh systolic **murmur** over the left interscapular area due to passage of blood in the coarctation.
- **Rosler's sign:** (on CXR)
Notching of the lower margins of the ribs, due to their erosion by the dilated collaterals.
- **Echocardiography:** is diagnostic.

TREATMENT

Medical:

- Prophylaxis for infective endocarditis.
- Treatment of complications.

Surgical:

- Balloon dilatation.

TETRALOGY OF FALLOT

IT CONSISTS OF

1. Pulmonary stenosis (infundibular type).
2. Ventricular septal defect (big).
3. Overriding aorta.
4. **S**light RV hypertrophy.

PATHOPHYSIOLOGY

Pulmonary stenosis results in:

1. Decreased pulmonary blood flow.
2. Increased RV pressure leading to:
 - a) *Right to left shunt of blood across the VSD.*
 - b) *RV enlargement, but only of slight degree, why? Because the presence of the big VSD prevents excessive rise of right ventricular pressure (the blood in the RV has 2 ways to leave the ventricle).*
3. Most of the deoxygenated blood passes from both ventricles into the overriding aorta leading to central cyanosis & only a small part passes through the PS to the lungs to get oxygenated.

CLINICAL PICTURE

Symptoms

1. **C**entral Cyanosis: Since birth.
2. **C**yanotic spells: “ Syncopal attacks ”
Due to spasm of the hypertrophied infundibulum forcing all the deoxygenated blood in the RV to the aorta leading to cerebral hypoxia, syncope, convulsions & more cyanosis.
3. **S**hortness of breath: exertional dyspnea.
4. **S**quatting position: improves the pulmonary blood flow & thus improves dyspnea because:
kinking the femoral arteries will increase the resistance to the blood flowing through the aorta, & thus forcing more blood to pass through the pulmonary artery to the lungs.
5. **S**tunted growth.

Signs

General:

1. Central cyanosis.
2. Clubbing of the fingers.

Cardiac:

A] Precordial examination:

- Slight RV enlargement (may be absent).

B] Auscultation:

1. Second sound:

- Single: aortic component only.
- Accentuated : due to:
 - *Increased aortic blood flow.*
 - *Aorta is anteriorly positioned.*

2. Systolic ejection murmur (maximum in 3rd left space): due to:
 - *Infundibular stenosis.*

COMPLICATIONS

1. **P**olycythemia, due to hypoxia.
2. **P**ulmonary TB due to lung oligemia.
3. **P**aradoxical embolism.

INVESTIGATIONS

1. CXR & screen:

1. Coeur en sabot:

- ◆ Dilated aorta.
- ◆ Small pulmonary artery (exaggerated waist).
- ◆ RV enlargement (uplifted apex).

2. Lung oligemia.

2. ECG:

- RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiography:

- ☐ Increased pressure in RV & decreased in pulmonary artery.
- ☐ Decreased O₂ tension in aorta.
- ☐ The catheter may pass from the RV to the aorta.
- ☐ Angiocardiography : will show the anomaly.

TREATMENT**Medical:** “ For cyanotic spells ”

- O₂ inhalation
- β -blockers or verapamil (they prevent & treat the attack by relaxing the infundibulum).

Surgical:

- Complete correction.
- Infundibular resection (Brock's operation).

TRIOLOGY OF FALLOT**IT CONSISTS OF**

1. Pulmonary stenosis (valvular type).
2. ASD.
3. **Marked** RV hypertrophy.

CLINICAL PICTURE

- Features of valvular pulmonary stenosis plus central cyanosis appearing 2 years after birth.

INVESTIGATIONS

1. **CXR:**
 - As PS.
2. **ECG:**
 - As PS.
3. **Echo and Doppler:**
 - Diagnostic.
4. **Catheterization:**
 - Diagnostic.

TREATMENT

- Medical:**
 - For prophylaxis and treatment of complications.
- Surgical:**
 - Complete surgical correction.

	Fallot's Tetralogy	Fallot's Triology
- Cyanosis	Since birth	Later on
- Squatting	Common	Absent
- Neck veins	Normal	Giant "A" wave
- Arachnodatyly & high arched palate	Absent	Common
- RV enlargement	Slight	Marked
- Pulmonary area	Resonant	Dull
- Thrill	Uncommon	Common
- Second HS	Single & accentuated	Widely split & weak
- Ejection click	Absent	Present
- Gallop	Absent	Common

DISEASES OF THE PERICARDIUM

1. Dry pericarditis.
2. Pericardial effusion.
3. Constrictive pericarditis.
4. Adhesive pericarditis.

DRY PERICARDITIS

ETIOLOGY

1. **I**diopathic (probably viral: Coxsackie B or Echo virus): the most common cause
2. **I**nfections: *Viral, Bacterial, TB.*
3. **I**nflammation: *FMF.*
4. **I**mmunological: *SLE, RA.*
5. **I**atrogenic: *INH, Hydralazine, Procainamide.*
6. **I**rradiation.
7. **R**enal failure.
8. **R**heumatic.
9. **P**ost-traumatic.
10. **P**ost-cardiotomy syndrome.
11. **M**yocardial infarction.
12. **M**alignant infiltration.

CLINICAL PICTURE

Symptoms

1. Symptoms of the CAUSE.
2. GENERAL symptoms: e.g.
 - *Fever, malaise, headache.*

3. LOCAL symptom: “ CHEST PAIN ”

- *It is due to inflammation of both the pericardium & the adjacent pleura.*
 - Site & Reference: Precordial & may radiate to the shoulders.
 - Character & Duration: Severe Stitching, Continuous.
 - Increases by: Inspiration & movements of chest wall.
 - Decreases by: Sitting up & leaning forward.
 - Associated symptoms: May be pleuritic chest pain.

Signs

1. Signs of the CAUSE.

2. GENERAL signs: e.g.

- *Fever.*

3. LOCAL sign: “ PERICARDIAL RUB ”

- *It is due to friction between the 2 layers of the inflamed pericardium.*
 - Site: Best heard over the Base of the heart & the Bare area.
 - Timing: Most obvious in systole, but may also be heard in diastole.
 - Character: Scratching
 - Increases by: Pressing the stethoscope against the chest wall.

Complications

Pericardial effusion.

INVESTIGATIONS

1 ECG:

- Elevated ST segment.
- Flat or inverted T waves.

2 Investigations of the cause.

DIFFERENTIAL DIAGNOSIS

1. DD of:

the causes of dry pericarditis.
2. DD of:

the causes of acute chest pain.
3. DD of:

the causes of elevated ST segment.

DD of elevated ST segment

	Acute pericarditis	AMI	Prinzmetal angina
<i>Elevated ST</i>	Concave upwards	Convex upwards	Flat
<i>Elevated ST</i>	In all leads	In some leads	In some leads
<i>Pathological Q wave</i>	Absent	Present	Absent
<i>Cardiac enzymes</i>	Normal	Elevated	Normal

TREATMENT

Symptomatic:

- Analgesics & Anti-inflammatory drugs for the pain.

Specific:

- Treatment of the cause.

The most important points in DRY PERICARDITIS

- Viral pericarditis is the most common cause (Coxsackie B & Echo).

• One of the most important causes of acute chest pain.

• Pericardial rub.

• One of 3 important causes of elevated ST segment in ECG.

PERICARDIAL EFFUSION

ETIOLOGY

1. **Seropericardium** **(ACCUMULATION OF EXUDATE)**
 - ▶ It occurs in all conditions that cause dry pericarditis especially:
 - TB: “the most common cause of pericardial effusion”.
 - Malignancy.
 - Viral.
 - Rheumatic.
 - ▶ Hemorrhagic pericarditis is a severe form of seropericardium with excessive RBCs:
 - TB.
 - Myocardial infarction.
 - Renal failure.
 - Malignancy.
2. **Pyopericardium** **(ACCUMULATION OF PUS)**
 - Severe inflammation of the pericardium due to:
Infection by pyogenic organisms, e.g. Staph. & Strept.
3. **Hydropericardium** **(ACCUMULATION OF TRANSUDATE)**
 - HHeart failure.
 - Hepatic failure.
 - Hypoproteinemia, e.g. Nephrotic syndrome.
 - Hypothyroidism.
4. **Hemopericardium** **(ACCUMULATION OF BLOOD)**
 - ▶ The pericardial fluid is frank blood:
 - Rupture of the heart: trauma or infarction.
 - Rupture of the coronaries: trauma during catheterization.
 - Rupture of: dissecting aneurysm of the aorta.
 - Hemorrhagic blood diseases.
5. **Chylopericardium** **(ACCUMULATION OF LYMPH)**
 - Obstruction: of thoracic duct.
 - Rupture: of thoracic duct.

PATHOPHYSIOLOGY

- The manifestations depend on:
 - Amount of fluid.
 - Rate of accumulation.
- Cardiac tamponade (severe compression) may occur in severe cases when:
 - The amount of fluid: *fills the pericardial reserve volume.*
 - The rate of accumulation: *exceeds the rate of stretch of the parietal pericardium.*
- The amount of fluid necessary to produce cardiac tamponade may be:
 - As small as 200 ml when the fluid collects rapidly.
 - Over 2000 ml when it collects slowly (*allowing adaptation of the pericardial sac*).
- Cardiac tamponade causes ↓ cardiac relaxation & ↓ cardiac filling:
 1. Systemic congestion: *due to stagnation of blood behind the heart.*
 2. Low CO.

CLINICAL PICTURE

Symptoms

1. Symptoms of: *Systemic congestion.*
2. Symptoms of: *Low CO.*
3. Precordial pain:
 - Dull aching pain: *due to stretch of the parietal pericardium.*
 - May be referred to: *the shoulder along the phrenic nerve.*
4. Pressure symptoms: *e.g.*
 - Dyspnea: *improves by sitting up & leaning forwards (prayer's position).*
 - Dysphagia: *due to compression of the oesophagus.*
5. Symptoms of the cause: *e.g. **TB toxemia.***

Signs

1. Signs of the CAUSE. e.g. TB.

2. GENERAL signs:

I. Decubitus: *Prayer's position.*

II. Signs of: *Systemic congestion:*

a) Neck veins:

- Congested pulsating neck veins.
- Kussmaul's sign: (inspiratory filling)
Due to failure of the right side of the heart to accept the increased venous return during inspiration.
- Attenuated or absent y descent.

b) Enlarged tender liver.

c) Ascites before oedema of lower limbs (ascites precox).

III. Signs of: *Low CO:*

a) Low SBP, weak pulse, pallor.

b) PULSUS PARADOXUS: (*↓ pulse volume during inspiration*)

- Description:

- It is a misnomer as it is an exaggeration of the normal pattern & not a paradox.
- In normal subjects, during inspiration, the lungs expand & accommodate more amount of blood, thus there will be a normal ↓ SBP (less than 10 mm Hg), and a ↓ pulse volume.
- In pericardial effusion, during inspiration, there will be an exaggerated ↓ SBP (more than 10 mmHg) & an exaggerated ↓ in pulse volume... * WHY ??

- Detection:

- It can be detected by palpation of the arterial pulse, or by detecting ↓ SBP (more than 10 mmHg) during inspiration using a sphygmomanometer.

- Value:

- It is an important clue to the presence of cardiac tamponade.

- **Explanation:** ¹

Since both ventricles (*in cardiac tamponade*) share a tight fixed incompressible covering, i.e., *the pericardial sac*, the inspiratory enlargement of the RV compresses and reduces LV volume; leftward bulging of the interventricular septum further reduces the LV cavity as the RV enlarges during inspiration. This will cause an exaggerated reduction in the LV stroke volume during inspiration.

- **Other causes of pulsus paradoxus include:**

- *Constrictive pericarditis.*
- *Restrictive cardiomyopathy.*
- *COPD.*
- *Severe bronchial asthma.*
- *Pulmonary embolism.*

3. **CARDIAC signs:**

A] **Inspection & palpation:**

- Apical pulsations: weak or absent.
- Precordial bulge: may occur in children.

B] **Percussion:**

- Dullness to the right border of the sternum.
- Dullness outside the apex.
- Dullness on the pulmonary area (shifting dullness).
- BARE AREA: ↑ size + stony dullness.
- EWART'S SIGN: dullness over the left lung base due to its compression collapse by the effusion.

C] **Auscultation:**

- Muffled heart sounds.

COMPLICATIONS

1. **Cardiac tamponade:** severe compression causing ↓ BP, ↑ JVP, HF or Shock.
2. **Constrictive pericarditis.**

Beck's Triad: ↓ BP, ↑ JVP, Muffled heart sounds.

¹ Harrison's principles of Internal Medicine "15th edition, 239 Pericardial disease"

INVESTIGATIONS

I. ECG:

- Low voltage.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).

II. IMAGING:

1. CXR:

- Symmetrical ↑ of cardiac shadow with smooth borders & loss of angulations (Flask-shaped).
- Cardiophrenic angle may be obtuse (Rotch's sign).
- Change of the shape of the heart with change of position.
- Change of the size of the heart from day to day.
- Double contour of the cardiac borders.
- Diminished cardiac pulsations under the screen.
- PULMONARY OLIGEMIA.

2. Echocardiography:

- The most sensitive test for detecting the presence of pericardial effusion & its severity.

3. Cardiac catheterization & angiocardiography: (not needed)

- Reveals a gap between the edge of the heart which contains the catheter and the peripheral cardiac shadow.
- Angiocardiography demonstrates the actual size of the heart inside the effusion.

III. OTHER INVESTIGATIONS FOR THE CAUSE:

- PERICARDIOCENTESIS: see later.
- Pericardial biopsy: e.g. for TB or malignancy.
- Serology: for SLE.
- Urea & Creatinine: for Renal failure.

PERICARDIOCENTESIS: (Aspiration of effusion)

- Indications of aspiration:

A] **Diagnostic:** "May be needed for diagnosis of the cause by analysis of the pericardial fluid"

a) Features of exudate & transudate effusions:

	TRANSUDATE	EXUDATE
Proteins	< 3 gm %	> 3 gm %
Fluid protein / serum protein	< 0.5	> 0.5
Specific gravity	< 1016	> 1016
LDH	< 200 IU / L	> 200 IU / L
Fluid LDH / serum LDH	< 0.6	> 0.6
Cells (WBCs)	< 1000 / cmm	> 1000 / cmm

NB Characteristics of TB effusion:

1. Exudate: rich in Lymphocytes & RBCs.

2. TB bacilli can be detected by:

- *Staining:* ZN stain.
- *Culture:* Lowenstein-Jensen medium or BACTEC.
- *Animal Innoculation:* Guinea pig.

b) Features of Pyopericardium:

- The fluid is **purulent** & contains many **pus cells**.

c) Features of Hemopericardium:

- The fluid is **bloody** & contains many **RBCs**.

d) Features of Chylopericardium:

- The fluid is **milky white** & contains many **fat**,
clears on addition of ether & stains orange with: Sudan III.

B] **Therapeutic:**

(see later).

- **Contraindications:**

- Bleeding tendency.
- Uncooperative patient.

- **Sites of aspiration:**

- From outside the apex.
- From the bare area.

- **Complications of aspiration:**

- Infection.
- Arrhythmias.
- Trauma: *myocardial rupture*, *pneumothorax*.

DIFFERENTIAL DIAGNOSIS

1. Congestive heart failure:

- Different types of dyspnea (*exertional, orthopnea, PND*).
- Oedema of LLs before ascites.
- No pulsus paradoxus.
- Heart is enlarged & there may be murmur & gallop.

2. Constrictive pericarditis:

- Prominent y descent.
- *Pericardial calcification:* on Chest x-ray.

3. Restrictive cardiomyopathy.

4. Liver cirrhosis & nephrotic syndrome.

5. DD of the cause of effusion.

TREATMENT

1. Treatment of the cause: **e.g. TB:**
 - Anti – TB drugs.
 - Corticosteroids: *to ↓ inflammation & prevent constrictive pericarditis.*

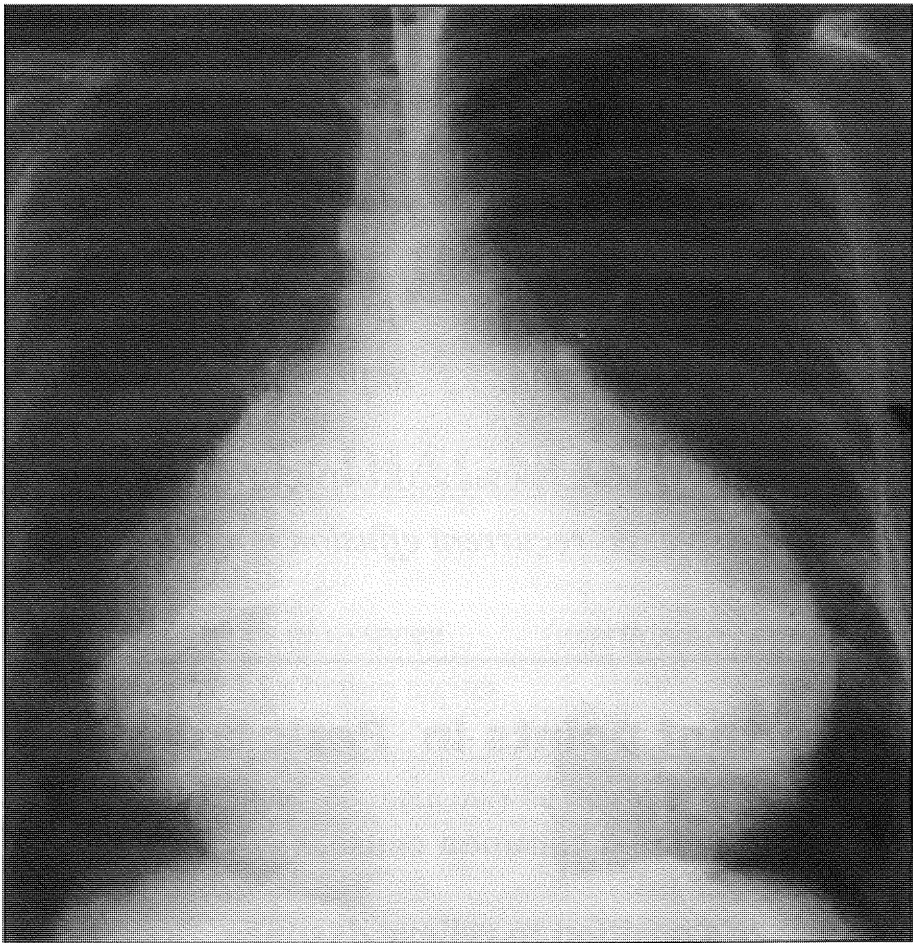
2. PERICARDIOCENTESIS: (Aspiration of effusion)
 - *In cardiac tamponade :* *to relieve dyspnea & compression.*
 - *In suppurative effusion :* *to drain pus & inject antibiotics.*
 - *In malignant effusion :* *to inject cytotoxic drugs.*

3. Pericardiodesis: (Pericardial sclerosis with Tetracycline)
 - *In malignant effusion:* *to prevent re-accumulation.*

4. Pericardiectomy or pericardio-pleural window:
 - *For massive & recurrent effusion not responding to medical ttt.*

Important points in Pericardial effusion

- ♥ TB is the most common cause.
- ♥ Prayer's position to relieve dyspnea.
- ♥ Pulsus paradoxus.
- ♥ Kussmaul's sign in neck veins.
- ♥ Echocardiography is the most important investigation.



CONSTRICTIVE PERICARDITIS

(Pick's disease)

ETIOLOGY

4 I

1. Idiopathic.
2. INFECTIONS:
 - Tuberculosis: the most common cause.
 - Bacterial, Viral, Fungal.
4. Immunologic: RA (but not RF).
5. Irradiation.

PATHOLOGY

- Dense fibrous tissue adheres the 2 layers of the pericardium together and constricts the heart. Frequently it undergoes calcification.

PATHOPHYSIOLOGY

- The rigid pericardium interferes with ventricular filling in late diastole. Ventricular filling is not reduced in early diastole, but it gets reduced abruptly when the elastic limit of the pericardium is reached. This is in contrast to pericardial effusion where ventricular filling is reduced all through diastole.

CLINICAL PICTURE

Symptoms

1. Symptoms of: Systemic congestion.
2. Symptoms of: Low CO.
3. Symptoms of the cause: e.g. TB toxemia.

Signs

1. Signs of the CAUSE. e.g. TB.

2. GENERAL signs:

I. Decubitus: No special decubitus.

II. Signs of: Systemic congestion:

a) Neck veins:

- Congested pulsating neck veins.
- Kussmaul's sign: (inspiratory filling)
Due to failure of the right side of the heart to accept the increased venous return during inspiration.
- Freidreich's sign: (diastolic collapse)
Prominent y descent due to rapid emptying of the congested neck veins in a short time after opening of the tricuspid valve.

b) Enlarged tender liver.

c) Ascites before oedema of lower limbs (ascites precox).

III. Signs of: Low CO:

a) Low SBP, weak pulse, pallor.

b) PULSUS PARADOXUS: in 30 % of the cases.

IV. AF: in 30 % of the cases.

3. CARDIAC signs:

A] Precordial examination:

- Apical pulsations: weak or absent.

B] Auscultation:

- Muffled heart sounds.
- **PERICARDIAL KNOCK:**
 - A sharp diastolic sound due to the sudden halting of the relaxing ventricles by the rigid pericardium.

INVESTIGATIONS

I. ECG:

- Low voltage.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- AF: in 30 % of the cases.

II. IMAGING:

1. CXR:

- Cardiac shadow is small.
- Calcification of the pericardium is diagnostic.
- Diminished cardiac pulsations under the screen.
- PULMONARY OLIGEMIA.

2. Echocardiography:

- Pericardial thickening.

3. Cardiac catheterization & angiocardiography:

- **Square root sign**: “Early diastolic dip followed by a plateau” in: RV & LV ventricular pressure tracings.

4. CT & MRI:

- Very sensitive in detecting: Pericardial thickening.

DIFFERENTIAL DIAGNOSIS

1. Congestive heart failure.
2. Pericardial effusion.
3. RESTRICTIVE CARDIOMYOPATHY.
4. Liver cirrhosis & nephrotic syndrome.
5. DD of the cause of constrictive pericarditis.

TREATMENT

1. Pericardiectomy:
 - Is the definitive therapy.
2. Treatment of the cause:
 - Anti – TB drugs (before & after pericardiectomy).

Important points in Constrictive Pericarditis

- ♥ TB is the most common cause.
- ♥ Pericardial knock.
- ♥ Calcification in chest x – ray.
- ♥ Restrictive cardiomyopathy is the main DD.
- ♥ CT or MRI is the most important investigation.

ADHESIVE PERICARDITIS

- * This condition may follow rheumatic fever which causes adhesions between the pericardium & the surrounding mediastinal structures & chest wall.
- * It has no significant hemodynamic effects & is rarely encountered.

RHEUMATIC FEVER

- An inflammatory disease that occurs as a sequel to pharyngeal infections by group A hemolytic streptococci.
- It involves principally the: joint, heart, CNS, skin & subcutaneous tissue.

ETIOLOGY

Probably an autoimmune process mediated through either:

1. Antigenic similarity:

Antibodies formed against streptococcal antigens react also with human tissue antigens since both are immunologically identical.

2. Altered antigenicity:

Streptococcal antigens bind to human tissue antigens rendering them antigenic and thus stimulating antibody formation against them.

PREDISPOSING FACTORS

- Age: Commonest between 5 & 15 years, Rare below 5 & above 25 years.
- Sex : Equal, except rheumatic chorea which is more common in females.
- FH: due to similar environment, (overcrowding & bad hygienic conditions).
- Recurrent streptococcal infections of the: pharynx & tonsils.

PATHOLOGY

“INFLAMMATION”

I. SITE:

1. Joints: synovial membrane.
2. Heart: pancarditis.
3. CNS.
4. Skin & subcutaneous tissue.
5. Small blood vessels.
6. Serous membranes: pleura & peritoneum.

II. TYPES:

1. Exudative Lesion:

- Affects mainly: serous membranes.
- Resolves completely: without residual effect.

2. Proliferative Lesion: “Aschoff nodule”:

- Affects mainly: the heart & the skin.
- Heals by fibrosis.
- Consists of:
 - *Central area of fibrinoid degeneration, THEN:*
 - *Macrophages, Lymphocytes & Aschoff's giant cells.*
 - *Outer layer of fibroblasts.*

CLINICAL PICTURE

1- MAJOR CRITERIA

1. ARTHRITIS

- Polyarticular affecting large joints: *e.g. knees, ankles, elbows.*
- It is fleeting or migrating in character, leaving the affected joint free.
HOWEVER, several joints may be affected simultaneously.
- The affected joint is painful, tender, red, hot, swollen with:
LIMITATION OF MOVEMENT.
- It is more common in adults, while in children, carditis is more common.
- There is a characteristic response to Salicylates.

2. CARDITIS

- It could be a pancarditis.
- It could be asymptomatic, Therefore:

Unless extra-cardiac manifestations as polyarthritis are associated, the condition will be missed. Then, later in life RHD may be recognized without definite history of RF.

- Rheumatic carditis may present with:

I. Endocarditis:

1. Mitral Valvulitis:

a) Mitral Regurge.

b) Mitral Stenosis:

- Carey Coomb's murmur: mid-diastolic murmur develops early in the acute stage due to swelling of the mitral cusps causing narrowing of the mitral orifice.
- Murmur of organic mitral stenosis: later on fibrosis occurs leading to permanent organic mitral stenosis, long after subsidence of the acute stage.

2. Aortic Valvulitis:

a) Aortic Regurge.

b) Aortic Stenosis.

II. Myocarditis:

1. Tachycardia disproportionate to the degree of fever.
2. Tic-Tac rhythm:
 S_1 & S_2 are similar due to loss of the muscular component of S_1 .
3. Cardiac enlargement & may be heart failure.
4. Cardiac arrhythmias & may be heart block.
5. Gallop rhythm.
6. Functional murmurs: due to ventricular dilatation.

III. Pericarditis:

- May be dry or with effusion.
- No constrictive pericarditis in rheumatic fever.

3. **CHOREA** (Sydenham's chorea) *

- Rapid involuntary movements (details in neurology).
- Never associated with arthritis.
- More common in females.

4. **SUBCUTANEOUS NODULES** (rare)

- Painless nontender small swellings:
 - a) Over bony prominences & tendons, e.g. dorsum of hands & feet.
 - b) Not adherent to the skin.

5. **ERYTHEMA MARGINATUM** (rare)

- Painless nontender erythematous (red) spots:
 - a) On the trunk & proximal extremities in crops.
 - b) The margin is serpiginous.
 - c) Gradually enlarge while the centers become pale, then: they disappear & reappear.

2- MINOR CRITERIA

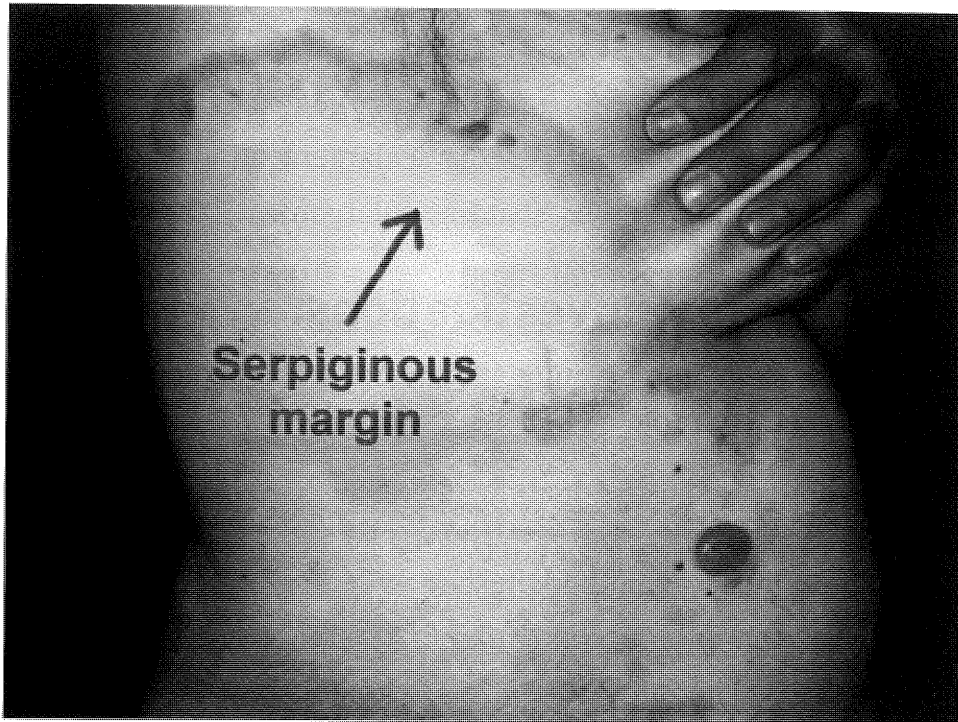
1. **P**revious rheumatic fever.
2. **P**resent fever.
3. **A**rthralgia.
4. **A**cute phase reactants: *leukocytosis*, $\uparrow$ *ESR*, $\uparrow$ *CRP*.
5. **P**R interval in ECG: *Prolonged*.

3- OTHER MANIFESTATIONS

1. Pleurisy.
2. Pneumonia.
3. Pain in the abdomen simulating acute abdomen.
4. Erythema nodosum.

* The British physician **Thomas Sydenham** (1624-1689), described chorea.

Erythema marginatum



Erythema nodosum

- **Important question:** **"Self – assessment"**
 "Mention other causes of erythema nodosum."

INVESTIGATIONS

A] Cardiac investigations:

1. ECG: cardiac enlargement, prolonged PR interval.
2. CXR: cardiac enlargement.
3. Echocardiography : cardiac enlargement, valve lesions.

B] Laboratory investigations:

1. Complete blood picture : anemia & leukocytosis.
2. C reactive protein (CRP): positive.
3. ESR: elevated.

4. Streptococcal antibody tests: *

a) Anti-streptolysin O titer (ASO)

- Titer higher than 250 todd units in adults & 400 todd units in children is indicative of recent streptococcal infection.
- Rising titer is more diagnostic.

b) Anti-streptozyme test (ASZ)

- Titer higher than 200 units / ml is present in nearly all patients with acute rheumatic fever.

5. Throat culture:

- May reveal group A streptococci.
- Less satisfactory than streptococcal antibody tests in diagnosing recent streptococcal infection.

DIAGNOSIS OF RHEUMATIC FEVER

“ Revised Jones Criteria ”

- The diagnosis is established by the presence of 2 points:
 1. Two major criteria or one major & 2 minor criteria.
 2. Evidence of recent streptococcal infection:

“Positive streptococcal antibody tests, or Positive throat culture”.

* Evidence of recent streptococcal infection.

DIFFERENTIAL DIAGNOSIS

- INFECTIVE ENDOCARDITIS.
- Causes of: fever in a cardiac patient.
- Other causes of: acute arthritis, carditis, chorea.
- Precipitating factors of: heart failure.

TREATMENT

A] Prophylactic

1. Primary (to prevent rheumatic fever)
 - Proper management of pharyngeal infections.
 - Tonsillectomy for chronically infected tonsils.
2. Secondary (to prevent recurrence of rheumatic fever; rheumatic activity)
 - Long acting penicillin: (Benzathine penicillin G)
 - 1.2 million units given monthly IM until the age of 25, or for 5 years after the last attack (whichever is longer), or for life.
 - Erythromycin: (for patients allergic to penicillin)
 - 250 mg / 12 h orally.

B] Therapeutic (only supportive)

- There is no specific treatment for RF & none of the measures will change the course of the attack. However, good supportive therapy can reduce mortality & morbidity.
1. Rest: until signs of inflammation disappear.
 2. Diet: light, poor in salt.

3. Antibiotics: (for eradication of streptococcal infection)

- **Benzathine penicillin G**: (Single IM injection)
1.2 million units (600.000 units for children below 10 years).
- **Erythromycin**: (for patients allergic to penicillin)
250 mg / 6 h orally for 10 days.

4. Anti-inflammatory drugs:

- These drugs relieve the manifestations of RF, BUT:
do not prevent chronic valvular lesions.

a) **Aspirin** (Acetyl salicylic acid): **For Arthritis**:

- 100 mg / kg / day orally in 6 divided doses until there is a satisfactory response (6 weeks).
- Antacids should be given to prevent gastritis.

b) **Corticosteroids**: **For Carditis**:

- **Prednisone**:
1 mg / kg / day orally in 4 divided doses for 4 weeks,
then gradual withdrawal in 4 weeks.
- **Aspirin**:
is given during withdrawal of steroids & continued for additional 4 weeks.
- **ACTH**:
1 ampoule IM twice / week in the last week.

5. Treatment of chorea: (Refer to neurology).

The most important points in Rheumatic fever

- ♥ Autoimmune etiology.
- ♥ The most common cause of valvular heart diseases, *especially MS*.
- ♥ Diagnosed according to: “Revised Jones criteria”.
- ♥ Investigations have a minor role in diagnosis compared to clinical picture.
- ♥ Treated by Aspirin for Arthritis, & by Corticosteroids for Carditis.

- **Important question:** **"Self – assessment"**
 "Mention important post – streptococcal sequelae."

- **Important question:** **"Self – assessment"**
 "Mention skin manifestations of rheumatic fever."

ENDOCARDITIS

DEFINITION

- Inflammation of the endocardial surface of the heart.

I. INFECTIVE ENDOCARDITIS:

1. According to the causative organism:

- Bacterial endocarditis.
- Non – bacterial endocarditis.

2. According to the clinical presentation:

- Subacute bacterial endocarditis (SBE):
 - Onset: Insidious.
 - Heart: Diseased heart.
 - Organism: Low virulence, e.g. Strept. viridans.
 - Prognosis: Low mortality.
 - Incidence: Common.
- Acute bacterial endocarditis (ABE):
 - Onset: Acute.
 - Heart: Diseased heart or normal heart.
 - Organism: High virulence, e.g. Staph. aureus.
 - Prognosis: High mortality.
 - Incidence: Rare.

II. NON – INFECTIVE ENDOCARDITIS:

e.g. *Rheumatic fever, SLE.*

INFECTIVE ENDOCARDITIS

ETIOLOGY

- For infective endocarditis to develop, there should be a combination of 2 factors:
Infection + underlying cardiac lesion.

I. INFECTION:

a) The causative organism:

- Gram-positive cocci:
 - Strept. viridans (*the most common organism*) “40 - 50 %”.
 - Strept. foecalis & Staph. aureus.
- Gram-negative bacilli:
 - **HACEK** group:
Hemophilus, Actinobacillus, Cardiobacterium, Eikarella, Kingella.
 - Klebsiella, Brucella, Pseudomonas, Proteus.
- Other organisms: Fungi, Rickettsiae, Chlamydia.

b) The route of infection:

- Dental procedures & upper resp. surgery: (*usually Strept. viridans*).
- GIT & Genitourinary procedures: (*usually Strept. foecalis*).
- Cardiac surgery & Catheterization: (*usually Staph. aureus*).

II. UNDERLYING CARDIAC LESION:

a) Valvular lesions:

- The mitral valve is most commonly affected followed by the aortic valve.
- The disease is not common in MS due to:
Low pressure gradient.

b) Congenital anomalies:

- Especially VSD, PDA & coarctation.
- The disease is not common in ASD due to:
Low pressure gradient.

c) Prosthetic valves: accounts for 10 – 20 % of the cases.

PATHOGENESIS

- Two factors are needed to increase the incidence of infective endocarditis:

I. High pressure gradient:

- It is common on mitral & aortic valves (*valves of the left side*).
- It is rare on tricuspid & pulmonary valves (*valves of the right side*).
- It is more common in MR than in MS.
- It is more common in VSD than in ASD.
- It is RARE in: AF.

II. Narrow orifice:

- It is more common in small VSD than in big VSD.

PATHOLOGY

- **Fibrin nests:**

Endothelial damage leads to deposition of platelets & fibrin (*fibrin nests*).

- **Vegetations:**

Blood-borne organisms colonise these nests, forming vegetations.

Therefore: vegetations are composed of bacteria surrounded by platelets & fibrin.

- **Embolization:**

Vegetations may detach leading to embolization (*septic emboli*).

- **Cardiac damage:**

Infection may result in damage & perforation of the cusps & chordae tendinae.

CLINICAL PICTURE

- Manifestations of the disease are due to:

1. *Toxemia.*
2. *Cardiac damage.*
3. *Embolization.*
4. *Immune complexes.*

A) General manifestations:

1. Temperature: Fever & general constitutional manifestations.
2. Pulse:
 - Tachycardia.
 - May be absent pulsations due to embolization.
3. General look: Marked pallor & toxic facies.
4. Eyes:
 - In the conjunctiva: Petichae (due to microemboli & capillaritis).
 - In the retina: Roth spots (oval hemorrhages + pale center).
 - SUDDEN BLINDNESS: due to embolism of the central retinal artery.
5. Hands & lower limbs:
 - Pale clubbing.
 - Osler's nodes: ¹
Small painful red tender raised, in the pulps of the fingers & toes.
They are due to: immune hyperplasia of the capillary endothelium.
 - Janeway's lesions: ²
Small painless red-blue nontender macules, in the palms & soles.
 - Splinter hemorrhages:
Linear hemorrhages under the nails (due to microemboli & capillaritis).

B) Cardiac manifestations:

1. UNDERLYING CARDIAC LESIÓN.
2. MURMURS:
 - o Change in the character of the already present murmurs.
 - o Development of new murmurs.
 - o Rupture cusps may give rise to a loud musical murmur:
"SEA GULL MURMUR"
3. HEART FAILURE.

¹ Sir **William Osler** (1849-1919), Canadian, had called attention to their diagnostic importance.

² The American physician **Edward G. Janeway** (1841- 1911), described the lesions.

C) Extra-cardiac manifestations:

1. Spleen:

- o Slightly enlarged soft & tender.
- o Stitching pain & splenic rub may occur due to embolization causing splenic infarction & perisplenitis.

2. Kidneys:

- o Renal infarction: causing *gross hematuria*.
- o Flea-bitten kidney: causing *microscopic hematuria*.
- o Acute glomerulonephritis immune – complex **GN**:
 - Nephritic syndrome or nephrotic syndrome.
 - Renal failure.

3. CNS:

“STROKE”

- o Embolic hemiplegia.
- o SAH: *due to rupture of mycotic cerebral aneurysm.*

4. Lungs:

- o Pulmonary embolism or pneumonia in cases of:
right-sided endocarditis (uncommon).

5. Joints:

- o Arthralgia or Arthritis.

COMPLICATIONS

1. Heart failure.
2. Renal failure.
3. Embolization.
4. Rupture mycotic aneurysm.
5. Complications of treatment.

SPECIAL FORMS

- **Endocarditis in IV drug abusers:**

- It may occur on a normal heart.
- Staph. aureus is most common & TV is most commonly affected.
- Pulmonary embolism & pneumonia are common.

- **Fungal endocarditis:**

- Often due to: Candida or Aspergillus.
- Of bad prognosis.
- Associated with negative blood culture.

INVESTIGATIONS

1. **Blood picture:**

- Anemia.
- *Leucocytosis*
- ↑ *ESR*
- ↑ *CRP*



Acute phase reactants

2. **BLOOD CULTURE:**

“the most important investigation”

- It is positive in most of the cases.
 - At least 3 blood samples are taken during fever.
 - The results are observed from 3 days up to 3 weeks.
 - Samples are cultured under aerobic & anerobic conditions.
 - If the patient was taking penicillin, penicillinase enzyme should be added.
 - **Culture-negative endocarditis may be due to:**
 - Prior use of antibiotics.
 - Infection with other organisms, e.g.: *hacek*, *fungi*, *rickettsiae*.

3. **ECHOCARDIOGRAPHY:**

a) Trans-thoracic echo:

- The underlying cardiac lesion.
- Any heart complication.
- VEGETATIONS, however, small vegetations may be missed.

b) Trans-oesophageal echo: “the most important investigation”

- Detects SMALL VEGETATIONS missed by trans-thoracic echo.

4. **Urine analysis:**

- Hematuria: Gross or microscopic.
- Proteinuria: and casts.

5. **Immunologic tests:**

- Circulating immune complexes: may be detected.
- Rheumatoid factor: may be positive.

DIFFERENTIAL DIAGNOSIS

- RHEUMATIC FEVER.
- Causes of: fever in a cardiac patient.
- Causes of: embolization.
- Causes of: GN.
- Causes of: tender splenomegaly.
- Precipitating factors of: heart failure.

DIAGNOSIS OF INFECTIVE ENDOCARDITIS

[MODIFIED DUKE CRITERIA]

Major criteria

1. Positive blood cultures (2 separate cultures for a typical endocarditis microorganism, such as *Streptococcus viridans*).
2. Positive echocardiographic findings: e.g. VEGETATIONS.
3. New valvular regurgitation:
 - o Change in the character of the already present murmurs.
 - o Development of new murmurs.

Minor criteria

1. Predisposition (history of IV drug use or congenital heart disease).
2. Fever $\geq 38^{\circ}\text{C}$.
3. Vascular phenomena (e.g. arterial emboli, intracranial hemorrhage, conjunctival hemorrhage).
4. Immunologic phenomena (e.g. GN, Osler nodes, Roth spots).
5. Positive blood cultures: without meeting the criteria above (major criteria).

Clinical criteria for definite infective endocarditis include:

- 2 majoror,
- 1 major and 3 minoror,
- 5 minor criteria.

TREATMENT

I. Prophylactic:

1. Correction of the underlying cardiac lesion.
2. Prophylactic antibiotics:
 - For dental procedures or upper respiratory surgery:
 - *Amoxycillin:* 3 gm orally, 1 hour before the procedure, and, 1.5 gm orally, 6 hours after the procedure.
 - For GIT or genitourinary procedures:
 - *Ampicillin:* 2 gm IV **plus** *Gentamycin:* 80 mg IV, ½ hour before the procedure, and, 6 hours after the procedure.
 - For cardiac surgery or catheterization:
 - *Cefotaxime:* 2 gm IV, 2 hours before the procedure, and, 1 gm / 6h for 4 doses after the procedure.

II. Therapeutic:

A) MEDICAL TREATMENT:

1. Specific treatment: **“Antibiotics”**
 - Once infective endocarditis is suspected, ttt should be started immediately without waiting the result of blood culture.
 - TTT should be continued for at least 4 – 6 weeks.
 - a) For Streptococci:
 - Penicillin G 20 – 30 million units / day IV for 4 weeks **plus**
 - Gentamycin 1 mg / kg every 8 hours IV for 4 weeks.
 - NB** For penicillin allergy, penicillin is replaced by Vancomycin 15 mg / kg every 12 hours IV.

b) For Staphylococci & gram-negative bacilli:

- Cloxacillin 2 gm / 4 hours IV *plus*
- Gentamycin 1 mg / kg every 8 hours IV.

c) For fungal endocarditis:

- Amphotericin B.

2. Symptomatic treatment:

- a) Rest: until clinical manifestations improve.
- b) Diet: light, poor in salt.
- c) TTT of complications.

B) SURGICAL TREATMENT:

- Replacement of the severely damaged valve.
- Replacement of the infected prosthesis.

ANATOMY OF THE CORONARIES

CORONARY ARTERIES

- There are **two main** coronary arteries which originate from the root of the ascending aorta:

1. The left coronary artery:

- It arises from the left sinus of Valsalva, passes forwards and to the left in the left atrioventricular groove for a short distance and then divides into **2 branches**:

a) The anterior descending artery:

It passes downwards in the anterior interventricular groove to the apex and then turns backwards to meet the posterior descending artery.

b) The circumflex artery:

It continues its course in the atrioventricular groove to meet the right coronary.

2. The right coronary artery:

- It arises from the right sinus of Valsalva, and then runs in the right atrio – ventricular groove to the posterior surface of the heart to meet the circumflex artery.

In the back of the heart it gives the posterior descending artery which runs downward in the posterior interventricular groove to meet the anterior descending artery.

PATTERNS OF CORONARY SUPPLY

1) Balanced circulation:

- The left coronary artery supplies:
The LA, LV and the anterior part of the interventricular septum.
- The right coronary supplies:
The RA, RV and the posterior part of the interventricular septum.

2) Right coronary predominance:

- The right coronary supplies also: *The posterior wall of the left ventricle.*

3) Left coronary predominance:

- The left coronary supplies also:
The posterior wall of the right ventricle & The posterior part of the septum.

ISCHEMIC HEART DISEASE

ETIOLOGY

1) Decreased myocardial oxygen supply:

a) Decreased quantity of coronary blood:

1. CORONARY ARTERY DISEASE:

i) CORONARY ATHEROSCLEROSIS (most common cause).

ii) OTHERS:

- **A**rteritis, e.g. PAN, SLE.
- **B**lood diseases, (Hypercoagulable) e.g. PRV, DIC.
- **C**ongenital anomalies.
- **D**issection.
- **E**mboli.
- *SPASM OF THE CORONARY* (good prognosis).

2. NON – CORONARY ARTERY DISEASE: (LOW CO)

- Any cause of low CO, especially: AS.

b) Decreased quality of coronary blood:

- Anemia.
- Cyanotic conditions (hypoxia).

2) Increased myocardial oxygen demand:

- ↑ myocardial contractility: e.g. Ventricular hypertrophy.
- ↑ preload: e.g. Hyperdynamic circulation.
- ↑ afterload: e.g. Systemic hypertension.
- ↑ HR: Tachycardia.
- SEVERE STRESS: (physical, mental).

ATHEROSCLEROSIS

1. What is atherosclerosis ??

- It is a process in which lipids, particularly cholesterol, are deposited in: the innermost layer of the artery (the intima) causing ATHEROMA.
- The atheroma causes gradual narrowing of the arterial lumen leading to: ISCHEMIA and necrosis of the tissues.
- The already present COLLATERALS open to bypass the narrow segment.

2. Lesions of atherosclerosis:

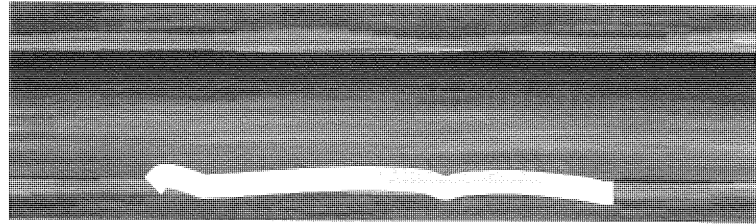
- Early Lesion (The fatty streak) *Contains 2 types of cells:*
 - o Foam cells : macrophages filled with lipids.
 - o T-lymphocytes: and some smooth muscle cells.
- Advanced lesion (The fibrous plaque)
 - o Thickened cap of dense CT.
 - o Large number of smooth muscle cells.
- Far advanced lesion (The complicated lesion):
 - o Degeneration , calcification.
 - o Ulcers, cracks, fissures.....and: **PLAQUE RUPTURE**.
 - o **Occlusion** of the lumen due to: plaque rupture or thrombosis.

3. Complications of atherosclerosis:

- Coronary Artery Disease (CAD).
- Cerebro – vascular Disease (CVD).
- Peripheral Arterial Disease (PAD).

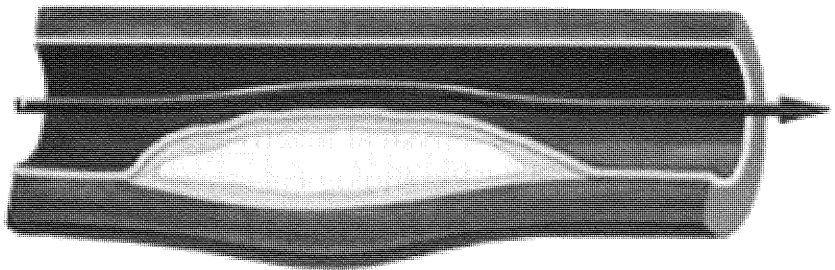
The lesions of atherosclerosis

1. Early lesion (The fatty streak)



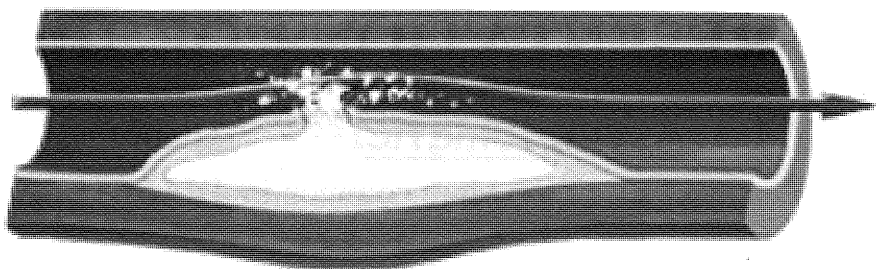
2. Advanced lesion (The fibrous plaque)

Plaque with
fibrous cap

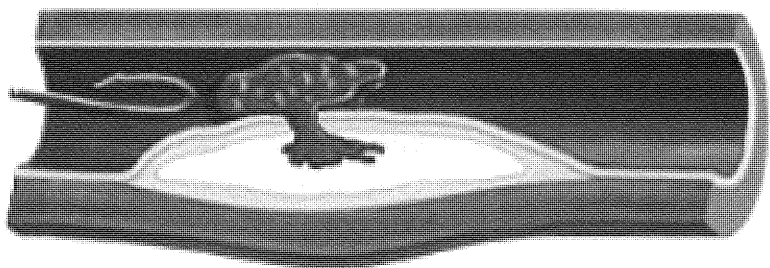


3. Far advanced lesion (The complicated lesion)

Cap
ruptures



Blood clot forms
around the rupture,
blocking the artery



4. Risk factors for atherosclerosis:

1. Non – modifiable:

- Age: usually above 40 years.
- Sex: male : female = 5 : 1.
- Type A personality : nervous, intellectual.
- Genetic factors: positive family history.

2. Modifiable:

a) High risk:

- DYSLIPIDEMIA: ↑ total cholesterol, ↑ LDL, ↓ HDL.
- Diabetes mellitus refer to DM.
- Hypertension: causes endothelial damage
- Cigarette smoking.

b) Less risk:

- Obesity.
- Diet: rich in saturated fat & cholesterol.
- Physical inactivity.
- Psychological stress.
- *Hyperuricemia.*
- *Homocysteinemia.*
- *Heavy alcohol intake.*
- CCPs.

PRESENTATIONS OF IHD

1. Asymptomatic: (silent myocardial ischemia)

- More common in DIABETES.

2. Symptomatic:

a) Typical presentation: (chest pain)

- Angina pectoris or acute myocardial infarction.

b) Atypical presentation:

- Congestive heart failure.
- Cerebrovascular stroke.
- Arrhythmias.
- Acute indigestion.
- Syncope.
- Sudden death.
- Peripheral embolism.
- Marked fatigue.

ANGINA PECTORIS

DEFINITION

- Attacks of chest pain, caused by myocardial ischemia, usually lasting for several minutes, and: **not resulting in myocardial necrosis.**

ETIOLOGY

- Angina occurs due to brief myocardial ischemia occurring when: **The myocardial oxygen demand exceeds the supply.**
- **What causes the pain ??**
 - When myocardial hypoxia occurs, metabolites accumulate & stimulate nerve endings in the myocardium to discharge impulses along **sympathetic fibres** to the lower cervical & upper thoracic segments of the spinal cord with subsequent radiation to the corresponding dermatomes, producing sensation of pain coming from these areas.
 - Pain may be **ABSENT** in **DIABETES** (Silent ischemia):
This is due to autonomic neuropathy.

CLINICAL PICTURE

1 **SYMPTOMS:** **“ CHEST PAIN ”**

- **Site & reference:** diffuse retrosternal, radiating to:
 - Shoulders, arms & forearms (especially the left).
 - Neck and lower jaw.
 - Less commonly: back or epigastrium.
- **Character & duration:**
 - Crushing, Compressing, Squeezing, Suffocating,
 - Several minutes (if > 20 min, AMI or UA should be considered).
- **Precipitated by:**
 - **S**tress (physical and emotional), **S**exual intercourse.
 - Cold weather, Heavy meals.
- **Relieved by:** rest, SL nitrates.
- **Associated symptoms:**
 - Dyspnea, palpitation, dizziness.
 - Sweating, nausea, vomiting.
 - ANGOR ANIMI: Sense of impending doom.

2 **SIGNS:** “ There may be no abnormality ”

- General signs: (during the attack)
 - Pallor, sweating.
 - Transient tachycardia and hypertension.
- Cardiac signs:
 - S₁: Weak.
 - S₂: Reversed splitting.
 - S₄: (due to decreased ventricular compliance by ischemia).
 - Signs of complications: e.g. systolic murmur of MR (pap. mus. dysfunction).

SEVERITY

“ 4 classes of angina according to severity ”

- CLASS I: Angina on marked activity, nothing occurs on ordinary activity.
- CLASS II: Slight limitation of physical activity on ordinary effort.
- CLASS III: Marked limitation of physical activity on less than ordinary effort.
- CLASS IV: Angina may be present even at rest.

TYPES

1. **Stable or Classical angina:** (typical form)

- The pain is relatively constant as regards severity, precipitating factors and relief.

2. **Unstable angina:**

- Worsening angina (crescendo angina): *increased frequency, severity or duration of a preexisting stable angina.*
- Angina starting to occur at **REST** (or with minimal exertion).
- Angina of **RECENT** onset (within 2 months).
- Angina **RESISTENT** to therapy.
- It carries a bad prognosis as 20% will pass to AMI within several months.
- It is considered an intermediate syndrome between stable angina and AMI.

3. **Variant angina:** (**Prinzmetal, Vasospastic angina**)

- Angina at rest: caused by SPASM of a coronary artery (*normal or atherosclerotic*), and is not precipitated by increased oxygen demand.
- ECG: *Transient ST elevation.*
- Ambulatory ECG: *Diagnostic.*
- Positive ergonavine provocative test: *Diagnostic.*
- Coronary angiography: *Normal or narrowed lumen.*

4. **Other forms of angina:** (**uncommon**)

- **Nocturnal:** it awakens the patient from sleep (due to ↑ sympathetic activity related to dreams).
- **Angina decubitus:** occurs on lying down and relieved by sitting (occurs in heart failure).
- **Angina of Lewis:** in cases of aortic regurge, it is nocturnal.

INVESTIGATIONS

1 Resting ECG:

- In between attacks: usually normal.
- During an attack:
 - ST segment: depressed (elevated in variant angina).
 - T wave: inverted.
 - ARRHYTHMIAS or HEART BLOCK.

2 Ambulatory ECG: * for diagnosis of variant angina.

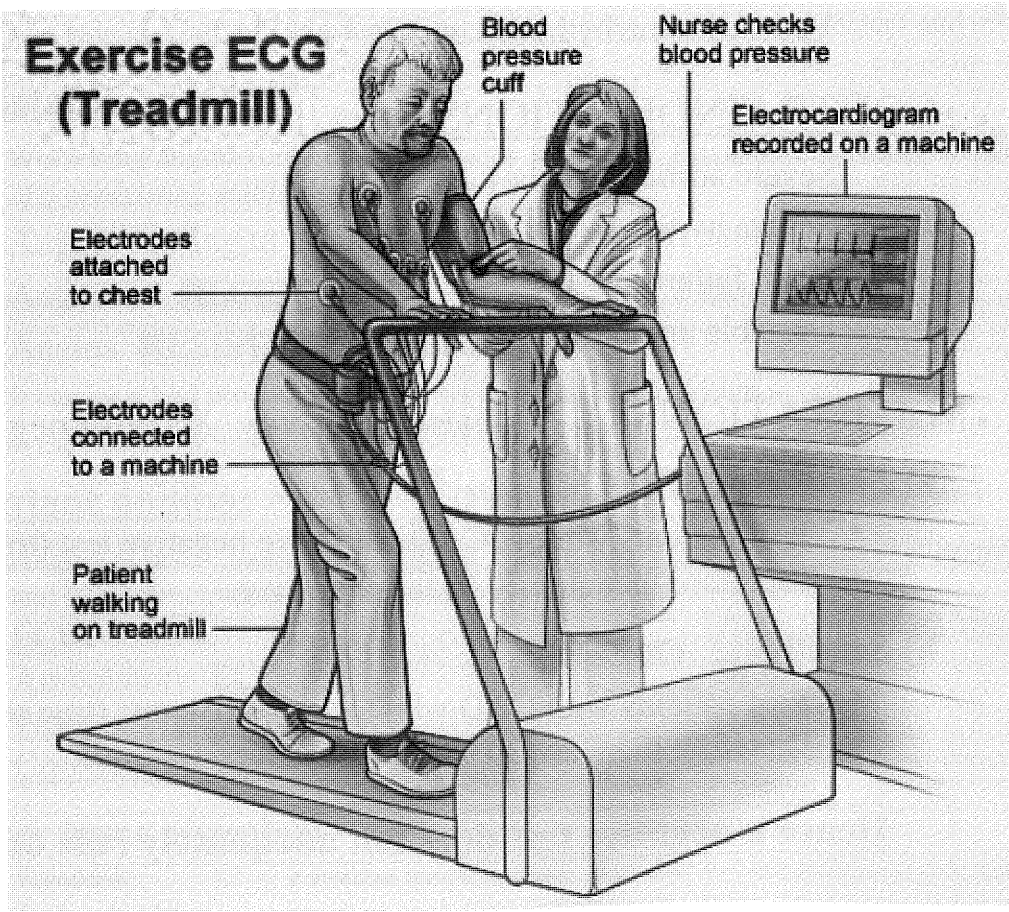
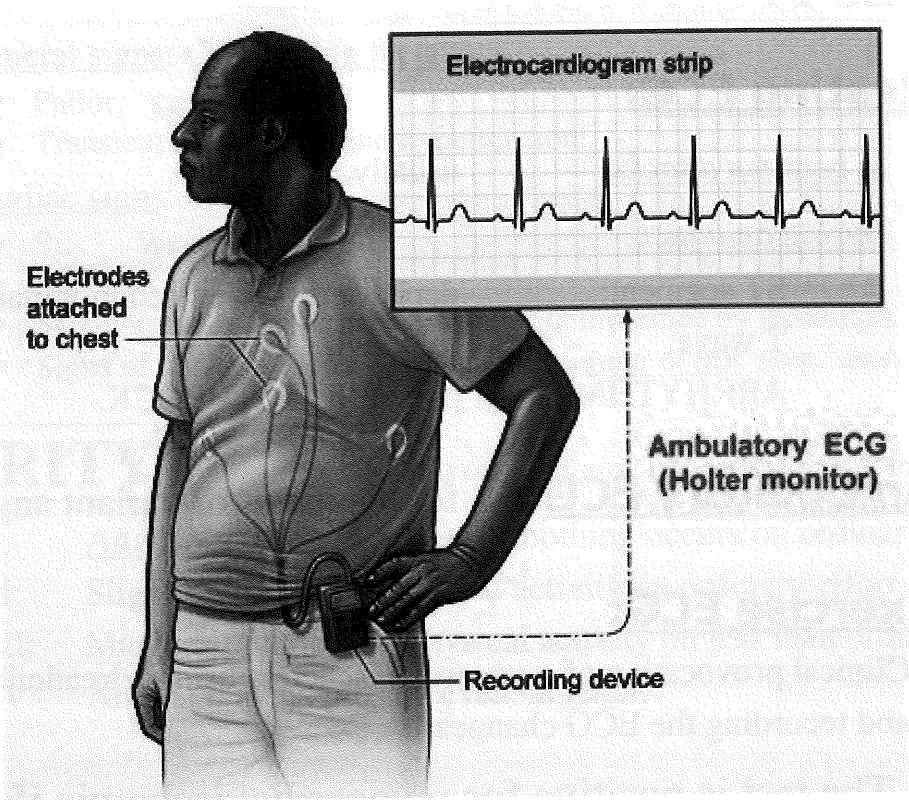
3 Exercise ECG:

“Clinical provocation of myocardial ischemia using a treadmill, and and recording the ECG changes ”

- The test is positive for myocardial ischemia if:
 - Typical anginal pain occurs.
 - ST segment depression occurs.
 - Ventricular arrhythmias occur.
- CONTRAINDICATIONS:
 - Severe aortic stenosis.
 - Severe hypertension.
 - Heart failure.
 - Unstable angina & recent MI (1 week).
- LIMITATIONS: “false positivity & false negativity”
 - a) **Specificity: (70 %)**
 - % of patients without CAD who have negative test (true negative).
 - Therefore there are 30 % false positive.
 - b) **Sensitivity: (70 %)**
 - % of patients with CAD who have positive test (true positive).
 - Therefore there are 30 % false negative.

Hence a negative test does not adequately rule out CAD

* Holter monitor: named after its inventor, Norman J. Holter (1914-1983), American.



4 **Isotopic studies:** (Radioactive Thallium – 201)

- **Thallium (201)** is taken up by healthy myocardium & not by the ischemic myocardium.
- Ischemia appears as a filling defect (**COLD SPOT**) at exercise & disappears after rest.

5 **Coronary arteriography:**

- It demonstrates the site and extent of coronary occlusion:

Indications:

- Angina which is: indicated for coronary revascularization.
- Angina which is: unstable, variant & post-infarction angina.
- Recurrent chest pain of uncertain etiology.

Complications:

- Myocardial infarction.
- Arrhythmias.
- Embolization.
- Arterial dissection.

6 **Recent imaging:**

- Multi – slice CT, Magnetic resonance coronary angiography (MR – CA).
- IVUS what is IVUS ??? 😊

7 **Stress echocardiography:**

- Methods:
 - Exercise.
 - IV dobutamine: *for people who are unable to exercise.*
- Result:
 - May show RWMA in ischemic areas (Hypokinesia).

8 **Biochemical screening:** (Blood tests)

- For identification of risk factors such as:
 - Dyslipidemia: lipids (TC, LDL, HDL, TG).
 - Hyperglycemia: glucose.
 - Hyperuricemia: uric acid.
 - Homocysteinemia: homocysteine.

DIFFERENTIAL DIAGNOSIS

- Causes of: acute chest pain

- Myocardium: Angina, AMI.
- Pericardium: Acute pericarditis.
- Endocardium: Mitral valve prolapse.
- Aorta: Dissecting aortic aneurysm.
- Pulmonary: Acute pulmonary embolism.
- Pain of cardiac neurosis.
- Acute pleurisy.
- Acute massive lung collapse & pneumothorax.
- Oesophageal pain: GORD.
- Peptic ulcer & cholecystitis.
- Herpetic pain (Herpes zoster).
- Musculo-skeletal disorders e.g. Tietze syndrome.

TREATMENT

1 DURING THE ATTACK

- Complete REST.
- SUBLINGUAL DRUGS:

<u>Nitrates:</u>	Nitroglycerin	0.5 mg	}	<u>Sublingually</u>
	Isosorbide dinitrate	5 mg		

These drugs act by:

- a) Dilating the coronaries.
- b) Reducing the work of the heart through: venodilator effect.

2 INBETWEEN THE ATTACKS

I. GENERAL MEASURES

a) TTT of the cause & correction of the risk factors:

- Cause: e.g. Vasculitis, AS, HTN.
- Risk factors: e.g. DYSLIPIDEMIA, * Diabetes, HTN.

b) Non – pharmacologic therapy:

نصائح

- Moderation of life: Avoid as possible the precipitating factors.
- Smoking: must be stopped.
- Diet control: Restriction of cholesterol & saturated fat.
- Physical activity: “REGULAR EXERCISE”
 - Improves the collateral coronary blood flow.
 - Improves the lipid profile.
 - ↓ the heart rate, BP & myocardial O₂ demands.
 - ↓ platelet aggregation, ↑ fibrinolytic activity.

II. SPECIFIC MEASURES

“Pharmacologic therapy”

دواء

AIM:

- To ↑ myocardial O₂ supply (dilate the coronary arteries),
- To ↓ myocardial O₂ consumption (↓ work of heart),

BY USING ANTI – ANGINAL DRUGS.

- To guard against the development of AMI (thrombosis),

BY USING ANTIPLATELETS and / or ANTICOAGULANTS.

* TTT of Dyslipidemia is mentioned later.

DRUGS:

1) ANTI – ANGINAL DRUGS: “Nitrates, βB , CCBs”

a) Nitrates:

*** Actions:**

- Dilate the coronary arteries.
- Reduce preload.

*** Preparations:**

- Nitroglycerine: 2.5 mg / 8 hours orally, or TP.
- Isosorbide dinitrate: 20 – 40 mg twice daily, or TP.
- Isosorbide mononitrate: 20 mg twice daily, or SR 100 mg, OD.

*** Side effects:**

- Severe headache.
- Tolerance (hyporesponsiveness):
 - o *Use the smallest effective dose.*
 - o *Allow a nitrate-free interval ($\simeq 8$ hs).*

b) β eta – blockers: (βB)

*** Actions:**

- $\downarrow$ HR & $\downarrow$ BP.
 - $\downarrow$ myocardial contractility.
- } $\downarrow$ **Cardiac work**

*** Preparations:**

- Non-selective: Propranolol, 60 mg / 8 hours.
- Cardio-selective: Atenolol, 50 – 100 mg / day.

*** Side effects:**

- *Chest:* Bronchospasm.
- *Heart:* Bradycardia, Hypotension, Heart block.
- *Others:* Fatigue, Reversible impotence,
Masking signs of hypoglycemia in diabetes.

c) Calcium channel blockers: (CCBs)

*** Actions:**

- Dilate the coronary arteries (extremely effective in VA).
- Reduce afterload.
- Reduce the myocardial contractility.

* **Preparations:**

- Verapamil 80 mg / 8 hours.
- Diltiazem 60 mg / 8 hours.
- Nifedipine 10 mg / 8 hours.
- Amlodipine: 5 mg / day.

* **Side effects:**

- Headache & flushing.
- Hypotension & aggravation of heart failure.
- REVERSIBLE OEDEMA OF LLs.
- Bradycardia: with *verapamil & diltiazem.*
- Tachycardia: with *nifedipine.*

Choice of drug therapy

1. Begin with nitrates, if there is poor response..... **THEN:**
2. For stable exertional angina: add β B or CCB to nitrates.
3. For recurrent angina on 2 drugs: apply triple therapy.
4. For variant angina: add CCB to nitrates.
5. For unstable angina: **heparin & aspirin** are most important.

2) ANTIPLATELETS & ANTICOAGULANTS:

- ***Aspirin*** (*low dose*): 75 – 300 mg / day (*single dose*).
- ***Dipyridamole***: 75 mg twice daily.
- ***Ticlopidine***: 250 mg twice daily.
- ***Clopidogrel***: 75 mg once daily.

They protect against AMI & death especially in patients with UA

- ***Heparin***:
 - IV heparin (with antiplatelets) is given in UA.

TTT OF DYSLIPIDEMIA

A) LIFE – STYLE MODIFICATION

- Diet control.
- Regular exercise.

B) DRUGS ¹

DRUG	ACTION	SIDE EFFECTS
<i>STATINS</i>	↓ cholesterol (↓ hepatic synthesis) ↓ LDL	Liver: ↑ enzymes Muscle: Myopathy and
<i>EZETIMIBE</i>	↓ cholesterol (↓ intestinal absorption) ↓ LDL	Liver: ↑ enzymes Muscle: Myopathy
SimvaSTATIN + EZETIMIBE = INEGY		
<i>Cholestyramine</i>	↓ cholesterol (Bile acid sequestrant) ↓ LDL	N, V, constipation
<i>FIBRATES</i>	↓ TG	↑ risk of severe myopathy with Statins
<i>Nicotinic acid</i>	↓ LDL, ↓TG ↑ HDL	Headache Diarrhea

¹ How would you deal with combined dyslipidemia ??

3 INVASIVE INTERVENTION

“Coronary revascularization”

INDICATIONS

1. General:
 - Angina which is not responding to medical treatment.
 - Post – infarction angina: *to improve the prognosis.*
2. Specific to the technique: *see below.*

TECHNIQUES

1. PCI (**P**ercutaneous **C**oronary **I**ntervention)

[PTCA + Stent (**P**ercutaneous **T**ransluminal **C**oronary **A**ngioplasty)]

- Introduction of a balloon to dilate the stenotic artery.
- Introduction of an intra – luminal **Stent** to maintain patency of the dilated artery.

o Indications:

- Stenosis of one or 2 vessels only (except left main coronary artery).
- Stenosis of bypass grafts following CABG.

o Complications:

- Complications of coronary arteriography (mentioned before).
- Acute coronary occlusion.
- Restenosis.

2. CABG *Surgical* (**C**oronary **A**rtery **B**ypass **G**raft)

- Grafting a segment of saphenous vein or an IMA between:
the ascending aorta & the coronary artery distal to the obstruction.

o Indications:

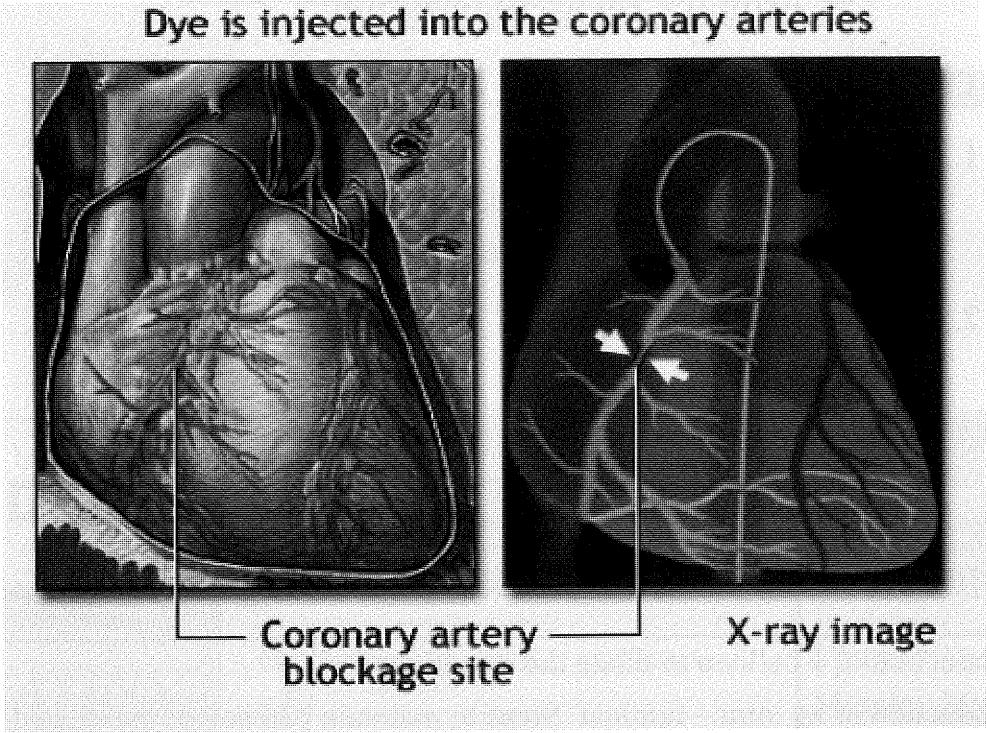
- Stenosis of 3 or more vessels.
- Stenosis of the left main coronary artery.

o Results:

- In 90 % of patients: *Excellent relief of angina.*
- In 10 % of patients: *Restenosis of the vein graft in the first year.*

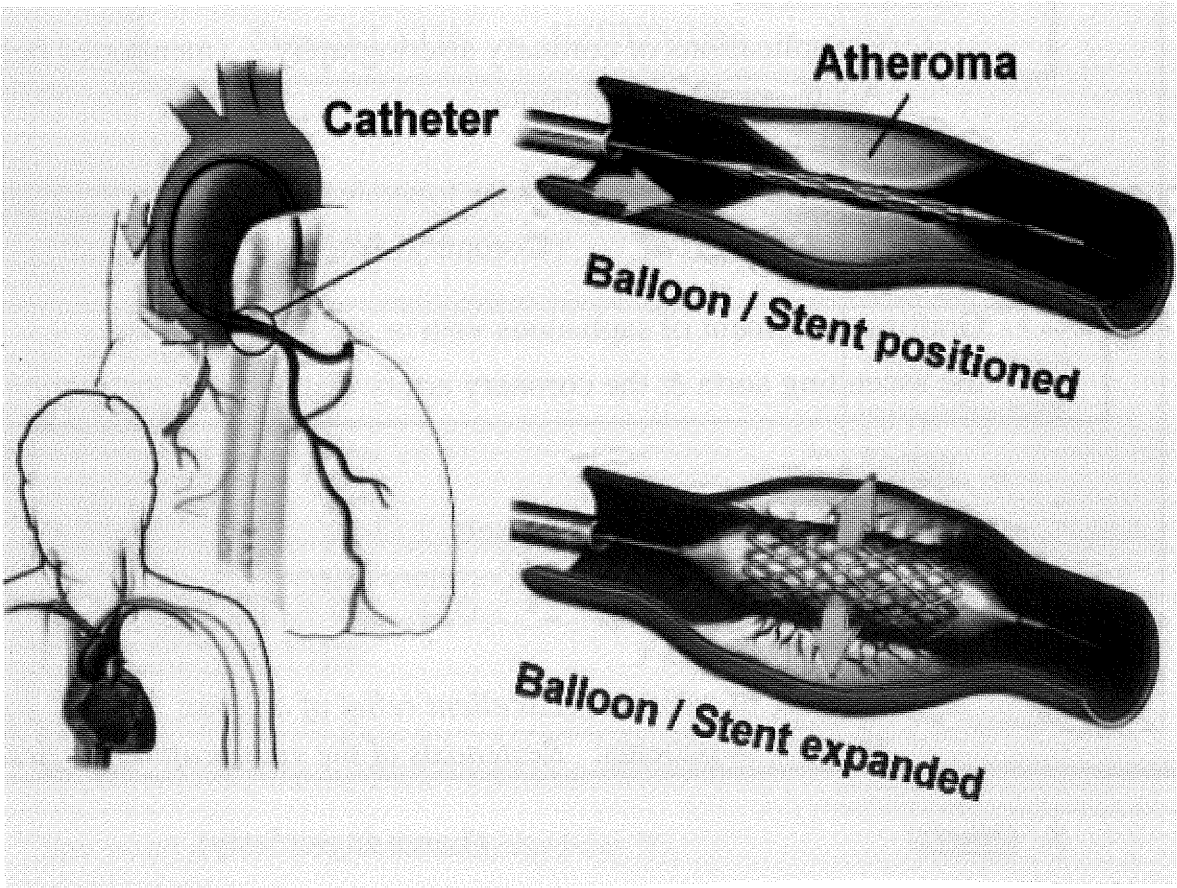
3. Others: (Laser angioplasty).

Diagnostic coronary angiography

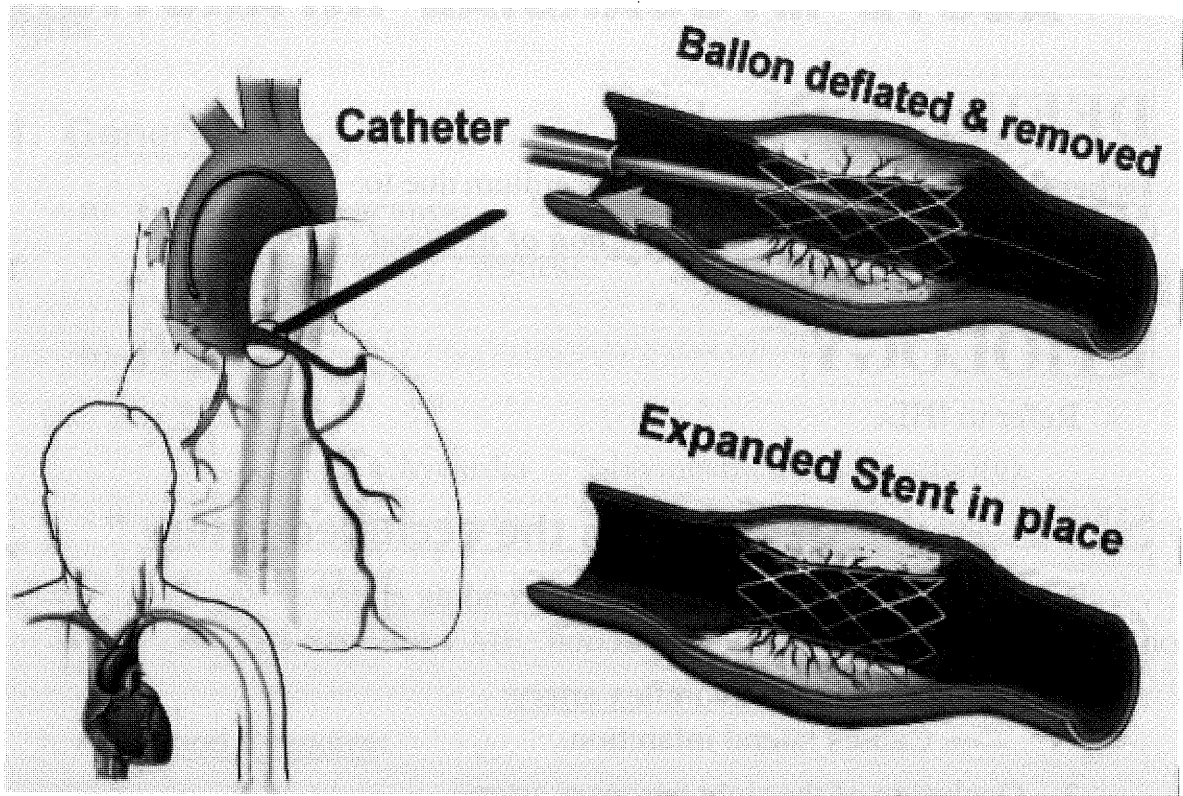


PTCA + Stent

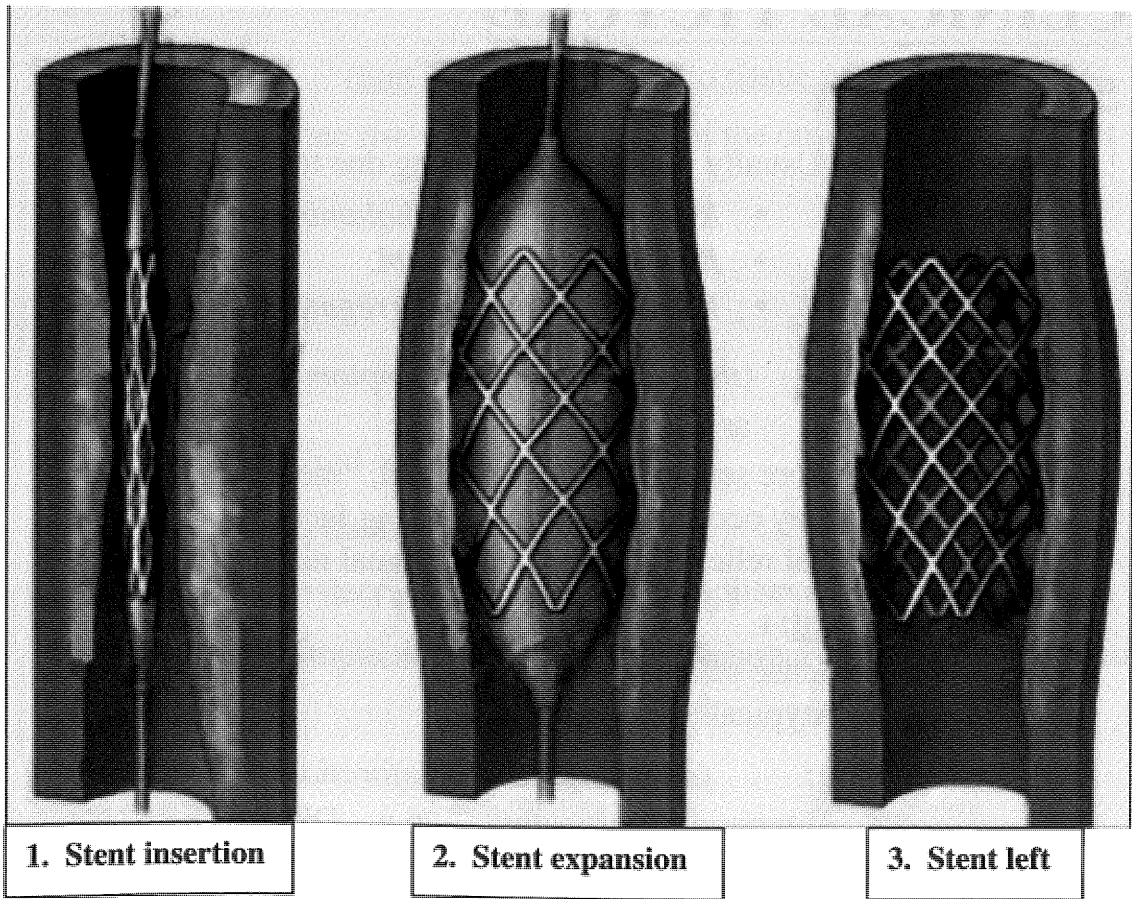
Step 1 & 2



Step 3



All 3 steps



ACUTE MYOCARDIAL INFARCTION

DEFINITION

Ischemic necrosis of part of the myocardium due to:

“ Sudden & persistent cessation of its blood supply ”

ETIOLOGY

- Refer to IHD.

SITE

“ Depends on the occluded artery ”

1. Occlusion of the left anterior descending artery:
 - Causes anterior infarction.
2. Occlusion of the circumflex artery:
 - Causes lateral infarction.
3. Occlusion of the right coronary artery:
 - Causes inferior infarction.

CLINICAL PICTURE

Symptoms

Onset: Usually in the early morning due to:

- *Rise in plasma catecholamines.*
- *Rise in plasma cortisol.*
- *Increased platelet aggregation.*

Pain: (*the main presenting symptom*)

a) **Typical:** as anginal pain, BUT:

- More severe, more prolonged, more radiating.
- May occur without precipitating factors.
- Not relieved by rest or sublingual nitrates.

b) **Atypical:**

- Sense of indigestion.
- Atypical location of pain.

c) **Absent:** (*silent myocardial infarction*)

- Autonomic neuropathy , e.g. DIABETES.
- ELDERLY PATIENTS.
- During anesthesia or in a transplanted heart.
- Disturbed consciousness level, heavy sedation, shock.

Signs

1. General:

a) General appearance:

- Pale, sweaty, restless.

b) Pulse:

- RATE:
 - Tachycardia (sympathetic hyperactivity).
 - Bradycardia (HB or ↑ vagal tone due to pain).
- RHYTHM: Arrhythmias.
- PULSE VOLUME: may be small due to LVF or shock.

c) Blood Pressure:

- Initial hypertension (sympathetic hyperactivity).
- Hypotension (LVF or shock or ↑ vagal tone due to pain).
- **Decapitated BP:** *marked drop of SBP with slight drop of DBP.*

d) Fever: (non specific response to tissue necrosis)

- Moderate rise within 1 – 2 days of the onset and may last for 1 week.

2. Cardiac:

- S₁: Weak.
- S₂: Reversed splitting.
- S₃: due to flabby myocardium.
- S₄: due to decreased ventricular compliance by infarction.
- Signs of complications: *e.g. systolic murmur of MR (pap. mus. rupture).*

COMPLICATIONS

1 Early Catastrophic Complications: *(first few days)*

1. **Sudden death:** *due to,*
 - VF.
 - Rupture of the weak myocardium.
 - Occlusion of the left main coronary artery.

2. **Cardiac arrhythmias:** *all types may occur,*
 - The most common are: extrasystoles.
 - The most serious are: VT & HB.

3. **Cardiac failure:**
 - Acute LVF.

4. **Shock:**
 - a) Cardiogenic:
 - Caused by massive infarction, leading to pump failure.
 - There is hypotension & tachycardia.
 - Prognosis: very bad.
 - b) Neurogenic:
 - Caused by severe pain, leading to vagal stimulation.
 - There is hypotension & bradycardia.
 - Prognosis: good.

5. **Myocardial rupture:**
 - Rupture of ventricular free wall → cardiac tamponade & death.
 - Rupture of the septum → acquired VSD.
 - Rupture of the papillary muscles → acute MR.

6. **Pericarditis:**
 - Usually dry, but effusion may develop.

2 **Late Complications** *(after several weeks)*

1. Post infarction angina & recurrent infarction:

- Due to affection of other coronaries.

2. Post infarction syndrome (Dressler's syndrome):

"Pleuropericarditis"

- Onset: developing weeks after infarction.
- Caused by: autoimmune response to damaged cardiac tissue.
- Responds to: corticosteroids.

3. Frozen shoulder (shoulder hand) syndrome:

"Pain & stiffness of left arm & shoulder"

- Onset: developing weeks after infarction.
- Caused by: reflex arteriolar spasm leading to ischemia & fibrosis.
- Responds to: corticosteroids & physiotherapy.

4. Thrombo – embolic complications:

- Mural thrombosis (in the LV) → systemic embolism.
- DVT (from prolonged recumbency) → pulmonary embolism.

5. Myocardial aneurysm:

- Clinical presentations:
 - Recurrent embolisation.
 - Recurrent arrhythmias.
 - Refractory HF.
 - Rupture of the aneurysm.
- On examination:
 - *Double apex.*
- Investigations:
 - *ECG:* persistent ST segment elevation > 6 weeks.
 - *Echo:* diagnostic.

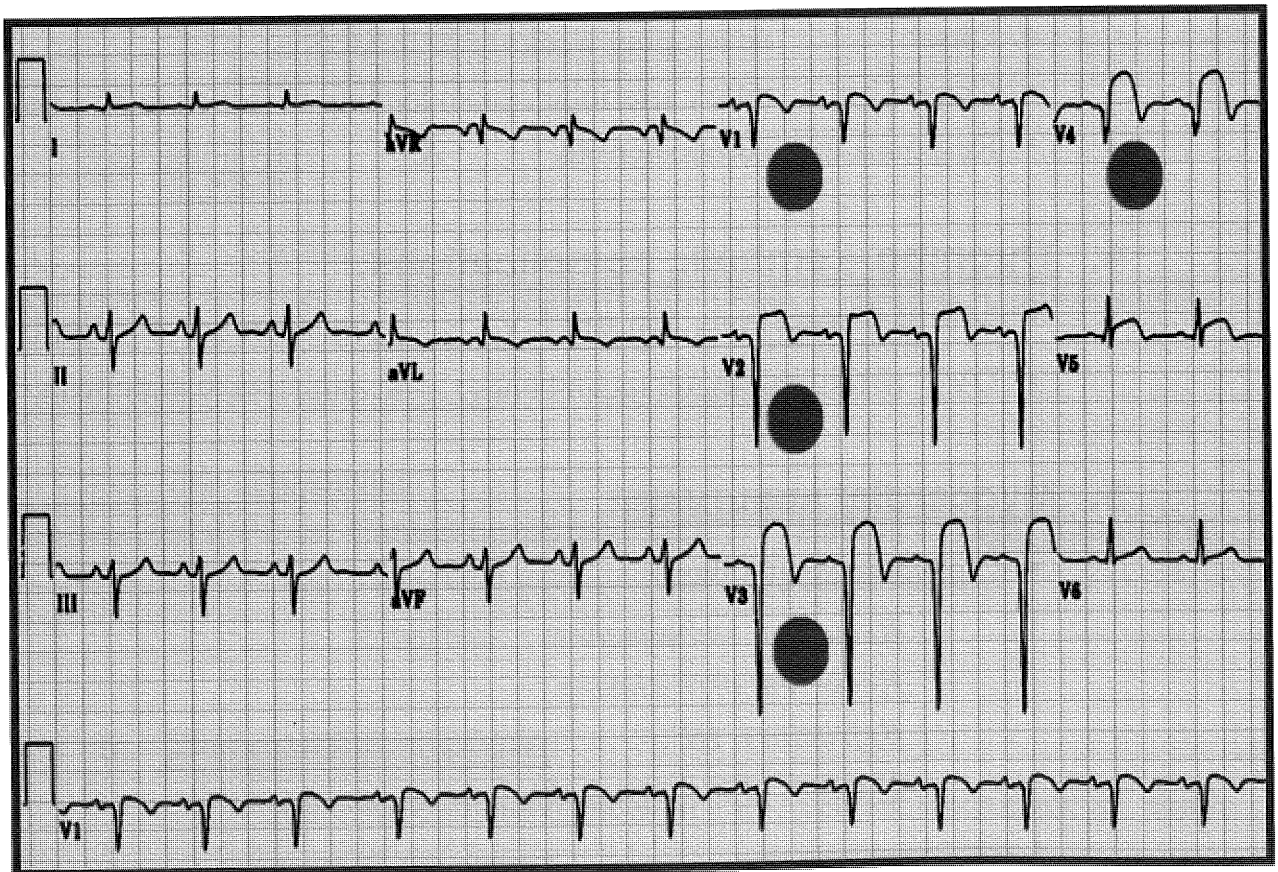
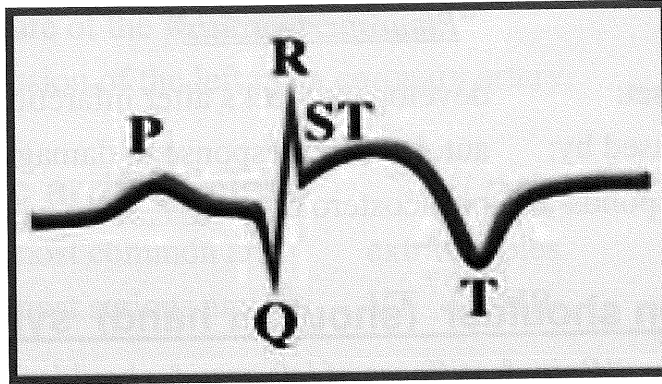
INVESTIGATIONS

1

ECG:

" Hyperacute T wave is the earliest sign "

- Inverted T wave = ischemia.
- Pathological Q wave = infarction.
- Elevated convex ST segment (injury pattern) = recent infarction.



Myocardial infarction

(Typical ECG pattern)

- Recent (elevated ST)
- Anteroseptal (V1 – V4)

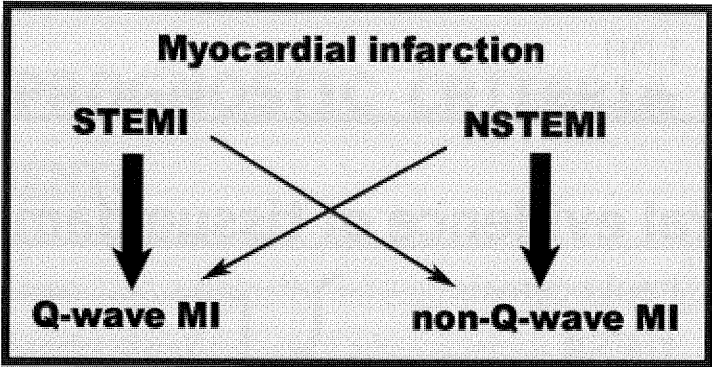
A normal ECG does not rule out acute myocardial infarction

According to ST segment, there are 2 types of MI:

- 1. **STEMI**: **ST** segment **E**levation **M**yocardial **I**nfarction
[confirmed by cardiac markers (enzymes)]
- 2. **NSTEMI**: **N**on **ST** segment **E**levation **M**yocardial **I**nfarction
[diagnosed by cardiac markers (enzymes)]

According to Q – wave, there are 2 types of MI:

- 1. **Q – wave MI**: “Previously called: Transmural MI”
[usually associated with: STEMI]
- 2. **Non Q – wave MI**: “Previously called: Subendocardial MI”
[usually associated with: NSTEMI]



According to affected leads, there are many sites of MI:

The constellation of leads with ↑ ST & pathological Q wave helps to identify what area of the heart is injured, & therefore helps predict the culprit artery:

Wall Affected	Leads Showing ST Segment Elevation	Suspected Culprit Artery
Septal	V ₁ , V ₂	LAD
Anterior	V ₃ , V ₄	LAD
Lateral	V ₅ , V ₆ , I, aVL	Circumflex
Anteroseptal	V ₁ , V ₂ , V ₃ , V ₄	LAD
Anterolateral	V ₃ , V ₄ , V ₅ , V ₆ , I, aVL	LAD or Circumflex
Extensive anterior	V ₁ , V ₂ , V ₃ , V ₄ , V ₅ , V ₆ , I, aVL	Left main coronary
Inferior	II, III, aVF	Right Coronary

2 CARDIAC MARKERS (Enzymes):

- Following AMI, the necrotic cardiac tissue will release several proteins:

a) Specific markers: “Typical enzymes”

- Troponins (I & T): ★
 - They start to rise within 4 – 8 hours & return to normal in 6 – 7 days.
- CK – MB: Creatine Kinase (Myocardial Band)
 - It is a specific subtype of the nonspecific CPK.
 - It starts to rise within 4 – 8 hours & return to normal in 2 – 3 days.

b) Non – specific markers: (old, not used)

- CPK.
- AST.
- LDH.
- Myoglobin.

3 General evidence of tissue damage:

- Leukocytosis
- ↑ ESR
- ↑ CRP (hs-CRP)

Acute phase reactants

4 Imaging:

a) Non-invasive:

1. Echocardiography:

- Akinesia of the infarcted area.
- Complications, e.g. MR, Myocardial aneurysm, Mural thrombosis.

2. Isotopic studies:

- Thallium imaging: infarcted area appears as a cold spot.
- Technitium imaging: infarcted area appears as a hot spot.

b) Invasive: (Coronary arteriography)

- To demonstrate: the site of the occluded vessel.
- To assess: the condition of the other coronaries.

c) Recent imaging:

- Multi – slice CT, MR – CA.
- IVUS.

DIFFERENTIAL DIAGNOSIS

- From all causes of acute chest pain (refer to angina).

PROGNOSIS

BAD PROGNOSTIC CRITERIA IN AMI

1. PATIENT:

- Age: old.
- Heart: originally unhealthy *weak* or *dilated* cardiac muscle.
- Associated risk factors of atherosclerosis & CAD: e.g. *DM*, *HTN*.

2. INFARCTION:

- Site: extensive anterior is worse than lateral & inferior.
- Size: the larger, the worse the prognosis.

3. MANAGEMENT:

- Delayed: and inability to apply early thrombolytic therapy.
- Ineffective: Failure of the patient's condition to stabilize during the first few days of management.

4. COMPLICATIONS:

- Development of severe complications: e.g. *cardiogenic shock*.
- Persistent elevation of the ST segment in ECG.

NB **ACUTE CORONARY SYNDROMES (ACS)**

1. Unstable angina.

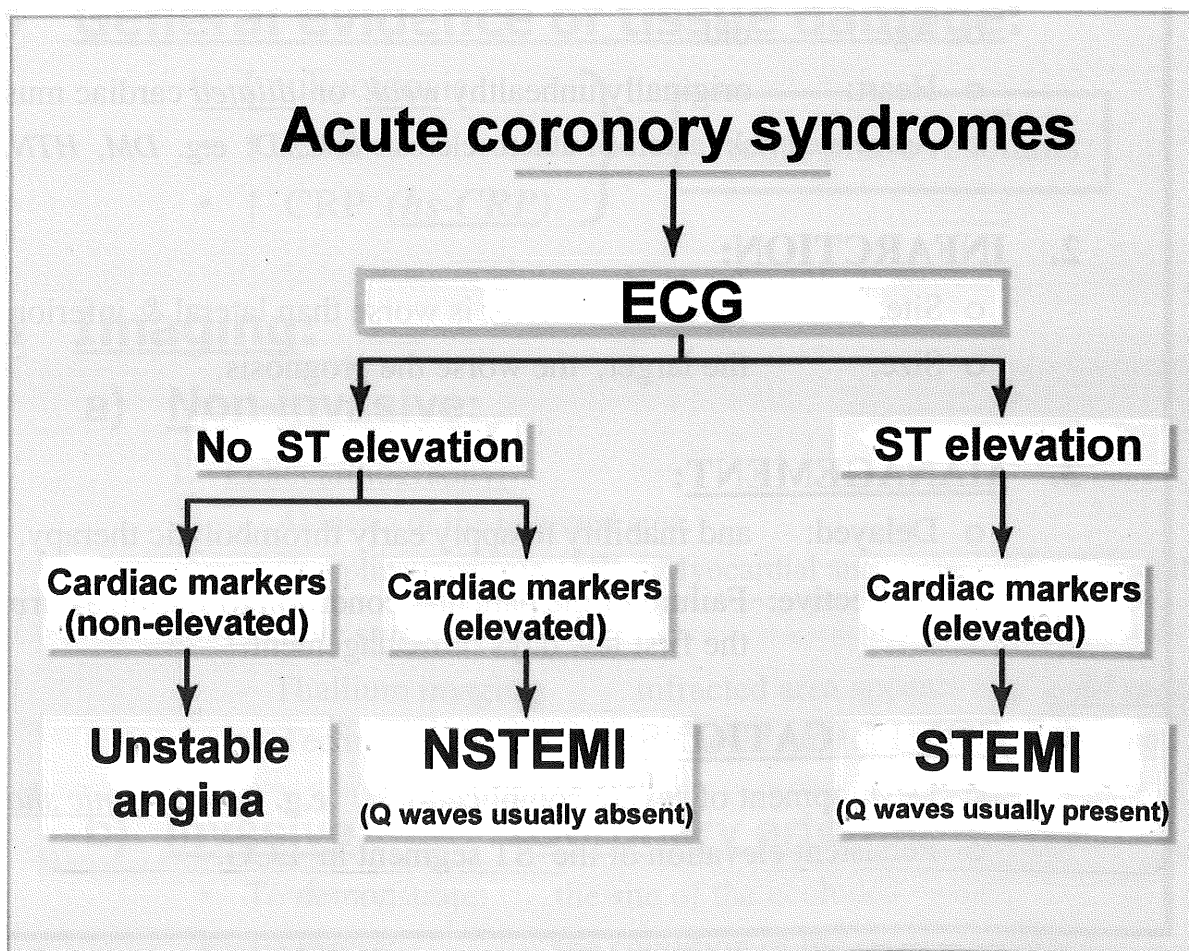
2. AMI:

- STEMI

[confirmed by cardiac markers (enzymes)]

- NSTEMI

[diagnosed by cardiac markers (enzymes)]



TREATMENT

1 PRE – HOSPITAL PHASE:

- a) Narcotic analgesics for pain:
 - Morphine: 10 mg IV or pethidine: 50 mg IV.
- b) Prophylaxis & Treatment of malignant ventricular arrhythmias:
 - Lidocaine: 100 mg IV.
- c) Treatment of increased vagal tone (*causing bradycardia*):
 - Atropine: 1 mg IV.
 - If bradycardia persists → pacing is needed.
- d) Prehospital coronary thrombolysis:
 - Important for early protection of the myocardium.
- e) Oxygen inhalation.

2 HOSPITAL PHASE:

“CCU”

1. General:

- Monitoring: continuous hemodynamic & ECG monitoring.
- Management of urgent complications, e.g.,
 - 1. *Fatal Ventricular arrhythmias:* *DC cardioversion.*
 - 2. *Heart block:* *Pacing.*
- Rest: Physical, Mental (sedatives, e.g. Diazepam 2 – 5 mg tds).
- Diet: Light frequent meals & stool softeners to avoid constipation.
- Oxygen inhalation.
- Control of associated DM:
 - o Diet control.
 - o Drugs: INSULIN (small doses of short acting).
 - o AVOID: HYPOGLYCEMIA.
- Prophylaxis against STRESS ULCER:
 - o Proton pump inhibitors (e.g. Omeprazole), or,
 - o H₂ receptor antagonists (e.g. Ranitidine).

2. **Refractory or continuous chest pain:**

- IV morphine or pethidine.
- IV Nitroglycerine (10 – 200 µg / min).
- IV or oral β-blockers.
- OXYGEN INHALATION.

3. **Angiotensin converting enzyme inhibitors:**

- They should be given within 24 hours to all patients post – MI to:
 - Reduce the progressive ventricular dilatation (**Remodeling**).
 - Reduce the incidence & severity of HF.

4. **Reperfusion:** (coronary thrombolysis) “to limit infarct size”

- **Fibrinolytic drugs:** e.g. Streptokinase, 1.5 million IU (IV infusion).
- **Timing:** as soon as possible; maximum benefit occurs within: **4 – 6** hours after the onset.
- **Result:** immediate dissolution of the thrombus, and, saving of the myocardium.
- **After Fibrinolytic drugs:**

Antipaletelet (aspirin) & Anticoagulant (Heparin), are given with & after fibrinolytic drugs.
- **Complications:**
 - Bleeding.
 - Reperfusion arrhythmias (e.g. **AIVR**).^{*}
- **Indications:**
 - Only in: STEMI.
 - Not in: NSTEMI (no benefit from thrombolytic therapy in NSTEMI).
- **Contraindications:**
 - Bleeding disorders.
 - Severe **H**ypertension.
 - **H**epatic failure.
 - **A**ctive peptic ulcer.
 - **A**ortic dissection.

* AIVR = Accelerated Idio – Ventricular Rhythm

5. Drugs that reduce the work of the heart:

- A) NITRATES: (IV nitroglycerine)
 - Given in the first days following AMI.
- B) β – BLOCKERS:
 - Given in the first days following AMI.

6. Anticoagulants:

- Following thrombolytic therapy.
- Thrombo-embolic complications.
- Intracardiac thrombi detected by echocardiography.

7. Antiplatelet drugs:

- **Aspirin** (low dose): 75 – 300 mg / day (single dose).
- **Dipyridamole**: 75 mg twice daily.
- **Ticlopidine**: 250 mg twice daily.
- **Clopidogrel**: 75 mg once daily.

8. Mechanical revascularization:

Methods:

- PCI: (PTCA + Stent).
- Surgery: (CABG).

Indications:

- In the patient who **does not respond** to thrombolytic therapy.
- Presence of a **contraindication** to thrombolytic therapy.
- **Following** thrombolytic therapy.

9. Treatment of early complications:

- Pump failure & shock.
- Arrhythmias.
- Others: “Surgical ttt” e.g.
 - Septal defect : closure of the defect.

3**POST – HOSPITAL PHASE:****“After Discharge”**

- Treatment of late complications:
 - Myocardial aneurysm: aneurysmectomy.
 - Frozen shoulder syndrome: corticosteroids & physiotherapy.
- REHABILITATION:
 - Gradual return to normal activity.
 - Regular exercise.
- Control of risk factors of atherosclerosis:

e.g. DM, HTN, Smoking, etc...
- Decrease the risk of post-infarction angina & re-infarction:
 - ASPIRIN.
 - β -blockers.
 - STATINS.

Diagnostic criteria of MI (WHO criteria)

1. **History:** Ischaemic chest pain > 20 minutes (Typical chest pain).
2. **ECG:** Changes in serial ECG tracings (Typical ECG).
3. **Enzymes:** $\uparrow$ Troponins, CK – MB, (Typical enzymes).

SYSTEMIC HYPERTENSION

DEFINITION

- Persistent elevation of the arterial blood pressure above 140/90 mm Hg in at least 2 measurements, on at least 2 separate occasions.
- PREHYPERTENSION:
Recently, BP from 120/80 mmHg to 139/89 mmHg is defined as "prehypertension." It identifies individuals at high risk of developing hypertension, (JNC 7) ¹
- WHITE COAT HYPERTENSION:
It is a phenomenon in which patients have elevated BP in a clinical setting **But not** when recorded at home.

It is believed that this is due to the *anxiety* some people experience during a clinic visit (Therefore: Ambulatory BP monitoring is required).

SEVERITY

	Systolic	Diastolic
Normal	Up to 140	Up to 90
Hypertension		
Stage 1 (mild)	140 — 160	90 — 100
Stage 2 (moderate)	160 — 180	100 — 110
Stage 3 (severe)	More than 180	More than 110

TYPES

1. Isolated Systolic Hypertension: ISH (rise of SBP only)
 - a) Increased LV stroke volume: Hyperdynamic circulation: e.g. AR.
 - b) Decreased aortic compliance: Aortic atherosclerosis.
2. Systolic & Diastolic Hypertension: (rise of SBP & DBP)
 - a) Essential (primary) hypertension.
 - b) Secondary hypertension.

¹ JNC 7 (The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure)

A) **Essential (primary) Hypertension:** (> 95 % of the cases)

Age: *It occurs in middle & old ages.*

ETIOLOGY *UNKNOWN*

Predisposing factors:

1. Genetic: runs in families.
2. Obesity.
3. Stress.
4. Salt sensitivity: 60 % of hypertensive patients are salt sensitive.
5. Smoking.

Theories for etiology:

1. **Activation of the RAAS**
 - ↑ secretion of renin which converts angiotensinogen to angiotensin I, and then to **Angiotensin II (AT II)** which causes hypertension through:
 - o AT II is a very potent VC causing ↑↑ PR.
 - o AT II stimulates aldosterone secretion → salt & water retention.
2. **Activation of the SNS**
 - ↑ secretion of catecholamines will cause VC & ↑↑ PR.
3. **Vascular hypertrophy: (& endothelial dysfunction)**
 - ↑ production of certain **growth factors** → vascular hypertrophy → ↑↑ PR & hypertension.
 - Hypertension → vascular endothelial injury (**endothelial dysfunction**), → further ↑↑ the production of growth factors → more hypertension.
4. **INSULIN RESISTANCE: (IR)**
 - Peripheral resistance to insulin occurs & → secondary hyperinsulinemia that elevates the BP through:
 - a) Increased Na reabsorption in the renal tubules.
 - b) Increased sympathetic activity.

B) **Secondary Hypertension:** (< 5 % of the cases)

- **C**ause – related hypertension.
- **C**urable hypertension.

ETIOLOGY

1. Renal causes:

a) Renal parenchymal:

- GN: Chronic & acute.
- Pyelonephritis: Chronic pyelonephritis.
- Collagen diseases: SLE & PAN.
- Metabolic: Diabetic nephropathy & Gouty nephropathy.

b) Renal vascular:

- Renal artery stenosis.

2. Endocrinal causes:

- **Pituitary:** Acromegaly.
- **Thyroid:** Hyperthyroidism, or Hypothyroidism.
- **Parathyroid:** Hyperparathyroidism.
- **Adrenal gland:**
 - *Adrenal medulla: Pheochromocytoma.*
 - *Adrenal cortex: Cushing's syndrome & Conn's syndrome.*

3. Neurological causes:

- ↑ ICT.
- Lesions of: *medulla & hypothalamus.*

4. Drugs:

- **C**atecholamines.
- **C**orticosteroids.
- **C**yclosporine.
- **C**CPs.
- **C**arbenoxolone.

5. Miscellaneous causes:

- Coarctation of the aorta.
- Toxemia of pregnancy.
- **P**olyarteritis nodosa.
- **P**olycythemia rubra vera.

Important question: "Self – assessment"

“Mention the clues that suggest Secondary Hypertension”.

CLINICAL PICTURE

1 **Benign Hypertension:**

- A mild form of HTN that runs a slow, long & usually asymptomatic course.

Symptoms

- Asymptomatic: *"the majority of patients"*
- Headache, tinnitus, dizziness, palpitation & epistaxis: *"a minority of patients"*

Signs

1. General:

- Pulse: $\uparrow$ pulse volume in: ISH.
- Blood pressure: persistent elevation above: 140/90.
- Fundus examination: reveals signs of HTN retinopathy.

2. Cardiac:

a) Precordial examination:

- Signs of LVH: with heaving apex.

b) Auscultation:

- S2: accentuated.
- Systolic ejection click.
- Systolic ejection murmur: due to relative AS.
- Soft early diastolic murmur: due to functional AR.
- S4: over the mitral area.

2 **Hypertensive urgency:**

- It is a severe form of HTN with rapid rise of BP: $> 220 / 120$ mm Hg.
- It is NOT ASSOCIATED with: target organ damage.

3 **Hypertensive emergency:**

- It is a severe form of HTN with rapid rise of BP: $> 220 / 120$ mm Hg.
- It is ASSOCIATED with: target organ damage.
- The organs primarily affected include:
 - CVS: Acute pulmonary oedema, AMI, Dissecting aortic aneurysm.
 - CNS: Hypertensive encephalopathy, Cerebral hemorrhage & SAH.
 - KIDNEY: Acute renal failure.
- There are 2 types:
 - Accelerated HTN: associated with retinopathy (hemorrhages & exudates).
 - Malignant HTN: associated with retinopathy (**PAPILLEDEMA**).

COMPLICATIONS

1. CVS:

1. ATHEROSCLEROSIS: *which may lead to,*
 - CAD (Angina & AMI).
 - CVD (TIA & Stroke).
 - PAD.
2. HEART: **LVF**, *due to,*
 - Pressure overload, coronary ischemia, diastolic dysfunction.
3. AORTA: Aortic dilatation & Aortic dissection.
4. BERNHEIM EFFECT:
 - Concentric hypertrophy of the LV may cause bulging of the septum in the RV cavity, leading to slight impairment of filling, and giant a wave in the neck veins.

2. CNS:

1. CEREBRAL ATHEROSCLEROSIS: (TIA & Stroke).
2. HEMORRHAGE: (Cerebral hemorrhage & SAH).
3. HYPERTENSIVE ENCEPHALOPATHY:

Mechanism:

- Attack of sudden severe ↑↑ of BP with generalized arteriolar spasm.
- Since the vasomotor tone of the cerebral arteries is weaker than that of the peripheral arteries, therefore the blood will rush to the cerebral circulation & causes cerebral oedema.

Clinical Picture:

- Manifestations of ↑↑ ICT: headache, vomiting, blurring of vision.
- Disturbed consciousness: Drowsiness then **coma** with:
 - o *Convulsions.*
 - o **NO SIGNS OF LATERALIZATION.**

4. Lacunar infarctions:

- Multiple small infarcts usually occurring in the corona radiata, internal capsule, cerebellum or basal ganglia.
- Clinically, they may be asymptomatic, or present with mild *motor or sensory manifestations, ataxia, dysarthria or extrapyramidal manifestations.*

3. **Renal:**

- Hypertensive nephropathy: *renal failure due to nephrosclerosis.*
- Hematuria & proteinuria.

4. **Retinopathy:** (4 grades)

- Grade I: Narrowing of the retinal arteries.
- Grade II: Marked sclerosis of the retinal arteries (silver wiring), Kinking of the veins at the arteriovenous crossings,
Plus: grade I.
- Grade III: Flame shaped hemorrhages & fluffy cotton exudates ...
Plus: grade II.
- Grade IV: Papilledema
Plus: grade III.

5. **Bleeding:**

- Epistaxis: *the most common.*
- Cerebral hemorrhage:..... *the most serious.*
- Other bleeding: (e.g. hematemesis or hemoptysis):... *uncommon.*

INVESTIGATIONS

1. **For the diagnosis of the cause:** (in secondary HTN)

1. Renal investigations: urine, renal functions, renal imaging.
2. Endocrinal investigations: lab tests for hormones, imaging.
3. Neurological investigations: CT, MRI brain.
4. Blood: CBC.

2. **For the diagnosis of complications:**

1. Cardiac investigations: CXR, ECG, Echocardiography.
2. Neurological investigations: CT, MRI brain.
3. Renal investigations: urine, renal functions, renal imaging.

3. **For detection of associated risk factors of atherosclerosis.**

TREATMENT

... Historical mystery ...

Believe it or not ...

In the year 1946 ...

“People with mild benign hypertension with levels up to 210 / 110 mm Hg need not be treated”

“There is a psychopathologic personality associated with hypertension”

1946 Textbook – Diseases of the Heart Friedberg

TREATMENT

1 Aim of treatment

- Complications: To avoid or ↓ the development of complications.
- BP goal:
 - To achieve & maintain BP < 140 / 90 mm Hg in hypertensive patients.
 - To achieve & maintain BP < 130 / 80 mm Hg in hypertensive patients with DM.

2 Plan of treatment

A) Primary (essential) Hypertension:

I. Non- pharmacological treatment.

II. Pharmacological treatment:

1. For Benign Hypertension.
2. For Hypertensive urgency.
3. For Hypertensive emergency.

B) Secondary Hypertension:

- Treatment of the cause.

I. Non-Pharmacological Treatment (Life style modification)

a) Dietary:

- Salt: must be restricted.
- WEIGHT REDUCTION IN OBESE PATIENTS.

b) Non-dietary:

- Smoking: must be stopped.
- REGULAR PHYSICAL ACTIVITY.

II. Pharmacological Treatment

1. FOR BENIGN HYPERTENSION (Stepped Care Therapy)

- ▶ The ttt passes in steps, if one step is not sufficient, we proceed to the next step:
 - **Step I:** **A**CE – I, or **β**-blocker or **C**alcium blocker, or **D**iuretic.
 - **Step II:** Combinations of 2 drugs of step I.
 - **Step III:** Combinations of 3 drugs of step I.
 - **Step IV:** Add α -adrenergic blocker or Hydralazine to step III drugs.
- ▶ The starting step:
 - In mild hypertension: start by step I.
 - In moderate hypertension: start by step II.
 - In severe hypertension: start by step III.

2. FOR HYPERTENSIVE URGENCY

- ▶ Aim of ttt is to lower the BP rapidly (**within 24 hours**) to stop complications.
However, too rapid ↓ of BP should be avoided as it may → organ hypoperfusion.
- ▶ TTT is urgently started using: diuretic, β B, VD, (**triple therapy**).

3. FOR HYPERTENSIVE EMERGENCY

- ▶ **General treatment for all emergencies**
 1. Hospitalization in an ICU.
 2. Rapid reduction of the BP to approximately 100 mm Hg diastolic: **within one hour** using rapidly acting drugs, **e.g.**
 - Diazoxide: 300 mg rapidly IV.
 - Hydralazine: 20 mg IV.
 - Na nitroprusside: 0.5 – 10 μ g/kg/min IV (**best in APE**).
 - Frusemide: 40 mg IV (**best in APE**).
 - α -methyl dopa: 250 mg IV.
 3. After control of BP, oral antihypertensive treatment should be started with drugs in step III.
- ▶ **Specific treatment for each emergency**
 - A) Hypertensive Encephalopathy:
 - Dehydrating measures: concentrated mannitol or glucose.
 - Anticonvulsants.
 - B) Other emergencies: refer to the related chapters.

Anti-hypertensive drugs

Group	Drug	Mode of action	Dose	Side effects
1. Diuretics	Refer to the chapter of “ Heart Failure”			
2. Anti-adrenergics				
a) Centrally acting	Alpha-methyl dopa (aldomet)	Central inhibition of the sympathetic system	250 - 1000 mg tds orally	- CAH - Hemolytic anemia
	Clonidine	Central inhibition of the sympathetic system	0.1 - 0.6 mg bid orally	- Postural hypotension - Rebound hypertension
b) Ganglion blockers	Trimethaphan	It blocks impulse transmission in the autonomic ganglia	1 - 6 mg / min IV	- Postural hypotension - Constipation & urinary retention
c) Nerve ending blockers	Guanethedine	It blocks the release of catecholamines from the adrenergic neurons	10 - 100 mg / day orally	- Postural hypotension
	Reserpine	It depletes the stores of catecholamines at the nerve endings	0.1 - 0.2 mg tds orally	- Depression
d) Alpha blockers	Prazocin	It blocks the alpha receptors causing VD	1 - 10 mg bid orally	- Sudden syncope - Tachycardia
e) Beta blockers	- Propranolol - Atenolol	- Reduction of CO	Refer to the chapter of “ angina ”	
f) Combined alpha & Beta blockers	Carvedilol	It blocks both alpha and Beta receptors	25 - 50 mg / day orally	- Same as β - blockers
3. Vasodilators	a) Hydralazine	Direct smooth muscle VD	10 - 50 mg tds orally or IV	- Tachycardia - Precipitates angina - Lupus-like syndrome
	b) Minoxidil	Direct smooth muscle VD	2.5 - 50 mg / day orally	- Tachycardia - Precipitates angina - Hirsutism
	c) Sodium nitroprusside	Direct smooth muscle VD	0.5-10 μ g /kg /min IV	- Thiocyanate poisoning
	d) Diazoxide	Direct smooth muscle VD	300 mg IV rapidly	- Hyperglycemia - Hyperuricemia

4. Angiotensin converting enzyme inhibitors (ACE – I)	<u>Captopril</u>	It blocks the conversion of Ag I to Ag II	<u>Captopril:</u> 12.5- 50 mg tds orally	- Dry cough - Proteinuria - Pancytopenia - Hyperkalemia - Renal failure if given in bilateral RAS
	<u>Ramipril</u>		<u>Ramipril:</u> 5- 10 mg/day	
5. Angiotensin receptor blockers (ARBs)	Losartan	It competitively Inhibits the binding of Ag II to its receptors	50 mg once daily orally	- Hyperkalemia - Renal failure if given in bilateral RAS
6. Calcium channel blockers (CCBs)	Amlodipine	It blocks the entry of Ca into the cells causing vasodilatation	5 - 10 mg / d orally	Refer to the chapter of “ angina ”
	Diltiazem		30 – 90 mg tds orally	
	Nifedipine		10 – 30 mg tds orally	
	Verapamil		80 – 160 mg tds orally	

Anti – Hypertensive drugs in certain situations

<i>Clinical situation</i>	<i>Preferred drug</i>	<i>Contraindicated drug</i>
A) Cardiovascular		
1. IHD	- CCBs (angina) - β B (angina & post-MI) - ACE – I (Post-MI)	- Hydralazine - Minoxidil
2. Heart failure	- Diuretics - ACE – I	- CCBs
B) Renal		
1. Renal failure	- Alpha-methyl dopa - Hydralazine	- Potassium-sparing diuretics
2. Bilateral RAS	_____	- ACE – I, ARBs
C) Others		
1. Bronchial asthma	_____	- β B
2. Diabetes mellitus	- ACE – I, ARBs, CCBs	- β B, Diuretics
3. Pregnancy	- Alpha-methyl dopa - Hydralazine	- Diuretics. - ACE – I.

- **Important question:** **“Self – assessment”**

“What is meant by: Refractory Hypertension” ??

PULMONARY HYPERTENSION

DEFINITION

Elevation of the pulmonary arterial pressure above 35 / 15 mmHg.

ETIOLOGY

1. Passive pulmonary hypertension:

- It is due to pulmonary congestion secondary to left sided HF.

2. Hyperdynamic pulmonary hypertension:

- It is due to increased pulmonary arterial blood flow.
- It occurs in congenital heart disease with left to right shunt, e.g. ASD & VSD.

3. Vasoconstrictive pulmonary hypertension:

- It is due to pulmonary arteriolar vasoconstriction.
- It occurs in:
 - Long standing passive pulmonary hypertension.
 - Long standing hyperdynamic pulmonary hypertension.
 - Long standing hypoxia, e.g. - COPD - Chronic interstitial lung disease.

4. Obliterative pulmonary hypertension:

- It is due to irreversible obliteration of the pulmonary arterioles.
- It occurs in:
 - Long standing vasoconstrictive pulmonary hypertension.
 - Recurrent minute pulmonary emboli.
 - Diseases of the pulmonary arteries, e.g.:
 - Pulmonary vasculitis, e.g. PAN.
 - Pulmonary bilharziasis.

5. Acute obstructive pulmonary hypertension:

- It is due to massive pulmonary embolism.

6. Idiopathic (primary) pulmonary hypertension:

- It occurs more commonly in middle-aged females with repeated pregnancies.
- It is of unknown etiology, but theories include:
 - Recurrent minute pulmonary emboli.
 - Pulmonary vasculitis.

CLINICAL PICTURE

Symptoms

1. Symptoms of low CO.
2. Symptoms of systemic congestion.
3. Symptoms of the cause.

Signs

General:

- Giant “a” wave in neck veins.

Cardiac:

A) Precordial examination:

- Pulmonary hypertension.
- RV enlargement.

B) Auscultation:

- Pulmonary hypertension.

INVESTIGATIONS

1. CXR:

- Dilated pulmonary artery.
- RV enlargement.

2. ECG:

- RA enlargement (P-pulmonale).
- RV enlargement.

3. Echocardiography:

- Estimation of the pulmonary arterial pressure.
- Detection of RV enlargement.

4. Cardiac catheterization:

- Estimation of the pulmonary arterial pressure.
- Detection of RV enlargement.

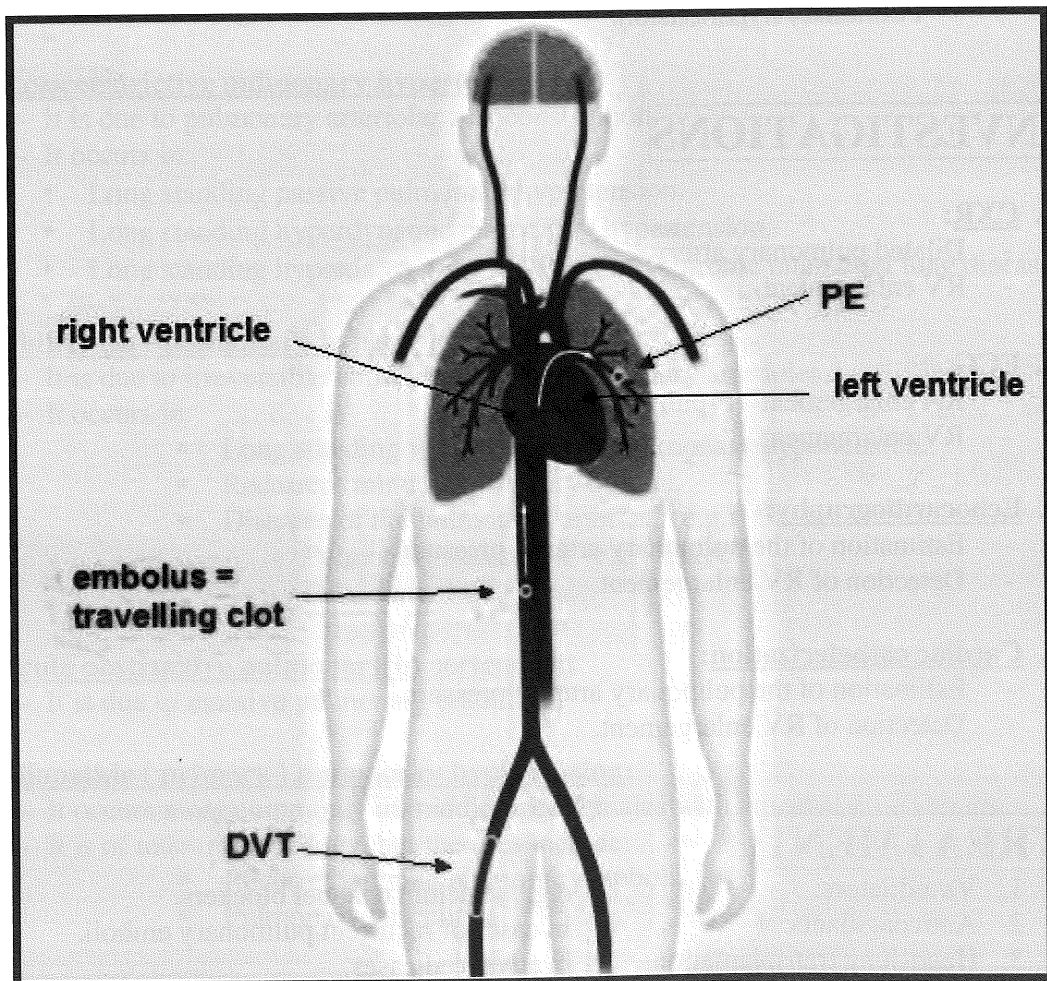
TREATMENT

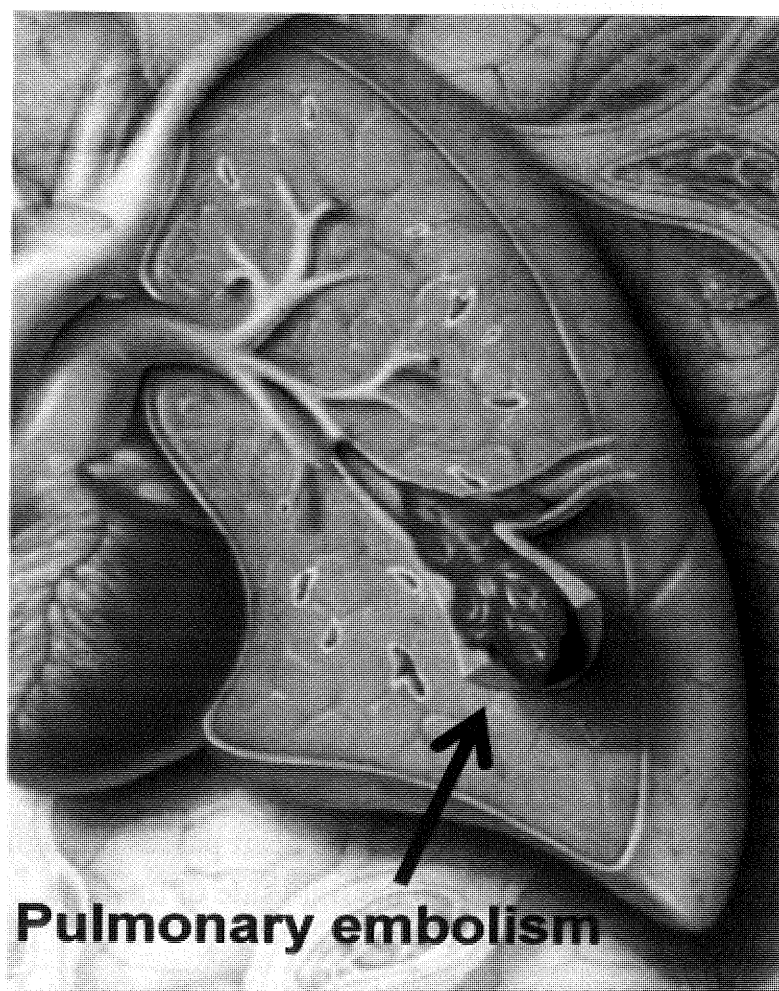
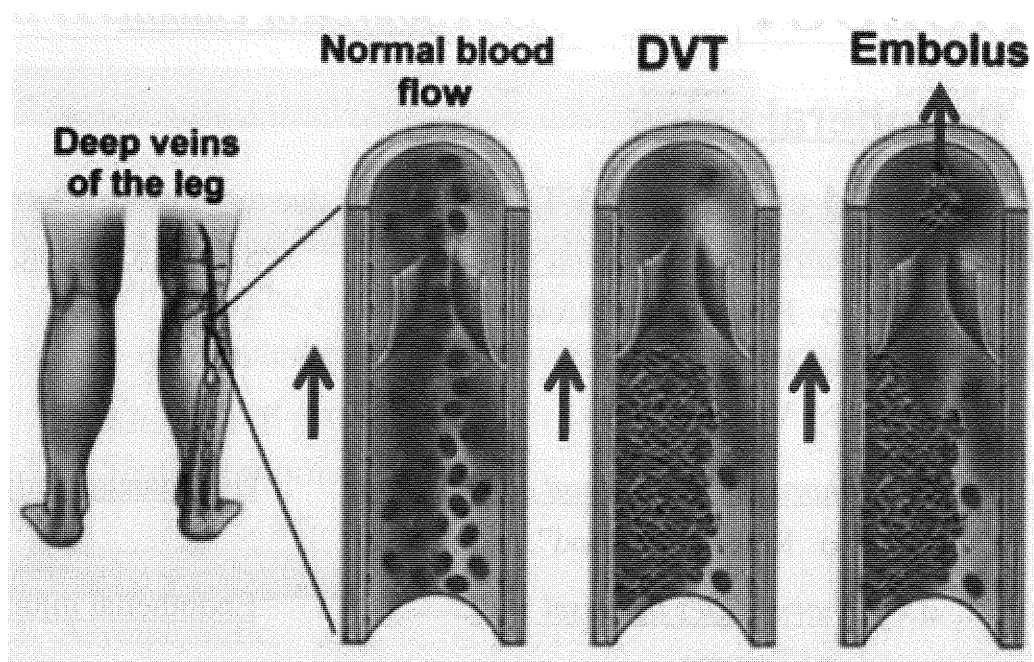
- | | |
|--------------------------------|---|
| 1. Vasodilators: | e.g. calcium channel blockers. |
| 2. Anticoagulants: | in cases of recurrent pulmonary emboli. |
| 3. Heart-lung transplantation: | in advanced cases. |

PULMONARY EMBOLISM

DEFINITION

- Sudden occlusion of a lung artery, usually due to a blood clot that travelled to the lung from a distant source in the circulation.
- Therefore , pulmonary embolism is not considered a disease; it is considered:
“ A complication of an existing thrombosis, *most commonly DVT of the leg*”.





ETIOLOGY

“Source of emboli”

1 Peripheral veins

A) Blood emboli: (DVT) (90 %), due to (Virchow's triad), *

1. Abnormalities in blood flow: “Slow circulation, Stasis”

- Prolonged bed rest.
- Heart failure.
- Varicose veins.

2. Abnormalities in blood composition: “Hypercoagulability”

- Refer to “Blood”.

3. Abnormalities in vessel wall: “Endothelial injury”

- Trauma.
- Inflammation.

B) Other (unusual) emboli: (very rare)

1. *Fat emboli.*
2. *Air emboli.*
3. *Parasitic emboli.*
4. *Malignant cell emboli.*

2 Right side of the heart (10 %)

1. RA thrombi: in cases of AF.
2. RV thrombi: in cases of RV infarction.
3. Right sided vegetations: in cases of infective endocarditis.

3 Paradoxical embolus (very rare)

- Embolus from the left side of the heart passes through a defect:
e.g. VSD or ASD to the right side of the heart.

* The German doctor **Rudolf Virchow** (1821-1902), described the triad.

CLINICAL PICTURE

I. Features of the source of embolism, e.g. *DVT*.

II. Features of pulmonary embolism vary according to:

- Whether the embolus is infected or not:
 - Infected embolus may lead to pneumonia & lung abscess.
- Number of emboli.
- Size of embolus.

A) Minute embolus

1. Asymptomatic.
2. Repeated showers of minute emboli cause occlusion of large number of pulmonary arterioles → thrombo-embolic pulmonary hypertension → → subacute cor pulmonale.

B) Medium-sized embolus → *pulmonary infarction (< 60 % occlusion)*

- Clinical picture:

1. General affection:
 - Fever & jaundice may occur.
 - Tachypnea & Tachycardia could be the only presenting features.
2. Pleural affection: (pleurisy)
 - Symptom: sudden stitching chest pain which ↑ by inspiration & cough.
 - Sign: there is pleural rub.
 - Complication: pleural effusion may develop.
3. Lung affection: *Acute dyspnea, cough & hemoptysis.*

- Differential Diagnosis:

- From other causes of: acute chest pain.
- From other causes of: acute dyspnea.
- From other causes of: hemoptysis.

C) **Big embolus**

1. **Acute massive pulmonary embolism:** (60 – 80 % occlusion)

- **Clinical picture:**

- **Acute Chest pain:** sudden severe retrosternal similar to that of AMI.
- **Acute Dyspnea:** and may be cyanosis.
- **Acute RVF:** rapid development of PH & acute RVF.
- **Shock:** cardiogenic.

- **Differential Diagnosis:**

From other causes of: acute ¹**chest pain**, acute ²**dyspnea**, acute ³**RVF** & ⁴**shock**,

- **Massive myocardial infarction.**
- **Massive lung collapse.**
- **Tension pneumothorax.**

2. **Sudden death.**

(> 80 % occlusion)

Did you know ...

Pulmonary embolism is one of the most common
causes of
unexplained sudden deaths

INVESTIGATIONS

I. INVESTIGATIONS for diagnosis of DVT

e.g.: Duplex ultrasound.

II. NON – IMAGING INVESTIGATIONS

1) ECG:

- RA enlargement (P – pulmonale).
- RV strain.
- RAD.
- S1 Q3 pattern.
- SINUS TACHYCARDIA.

2) Laboratory tests:

- Arterial blood gases: *Hypoxia & Hypocapnea.*
- Elevated serum LDH.
- Elevated serum indirect bilirubin.
- General evidence of tissue damage:
 - Leukocytosis.
 - Raised ESR.
 - Positive CRP.

Acute phase reactants

• PLASMA D – DIMER:

- It is a blood test that measures a substance in the blood that is released when a clot breaks up, **i.e.** it reflects the breakdown of fibrin by plasmin (*endogenous thrombolysis*).
- It is positive in more than 90 % of patients, THEREFORE:
A normal test makes pulmonary embolism not likely.

III. **IMAGING INVESTIGATIONS**

1) **CXR**

- Wedge shaped opacity.
- Linear opacity.
- Pleural effusion.

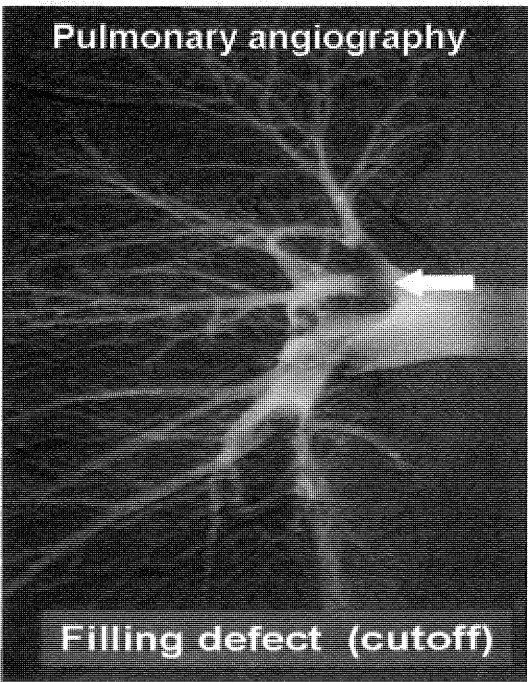
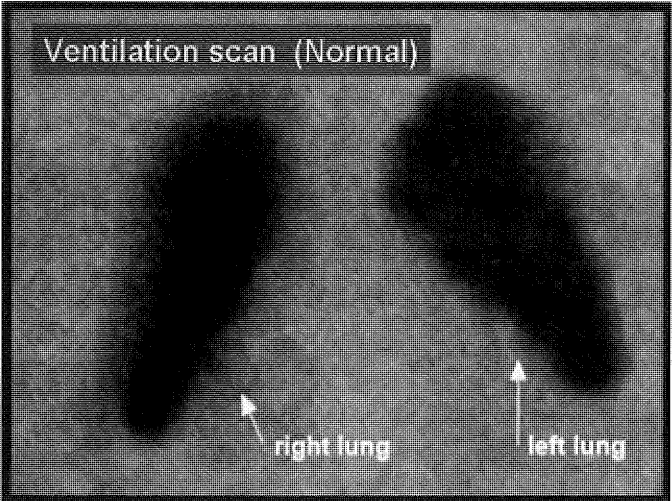
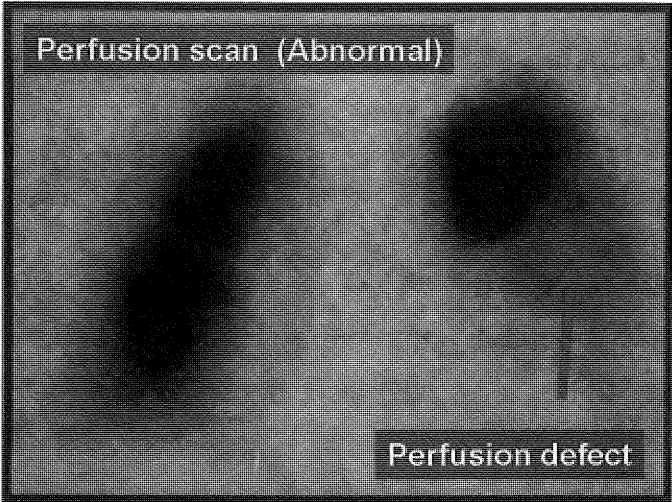
2) **Lung scanning** “*Ventilation – perfusion scan*”

- A test that uses a *radioactive material* to see if air and blood are flowing normally to areas of the lung.
- A lung ventilation/perfusion scan (V / Q scan) is actually *two tests* performed simultaneously:
 - **Perfusion scan:**
 - It is performed by injecting *radioactive albumin* into a vein.
 - The lungs are then scanned to detect the location of the radioactive particles as blood flows through the lungs.
 - *It measures the supply of blood through the lungs.*
 - Pulmonary embolism appears as a perfusion defect (*cold spot*).
 - **Ventilation scan:**
 - It is performed by scanning the lungs while the patient inhales *radioactive gas* (Xenon).
 - *It measures the ability of air to reach all portions of the lungs.*
 - Pulmonary embolism does not affect the ventilation scan.

Pulmonary embolism causes striking V / Q mismatch

3) **Pulmonary angiography**

- Accurate, BUT invasive.
- Positive results consist of: a *filling defect* or sharp *cutoff* of the affected artery caused by the embolus.



4) Spiral (Helical) CT scan

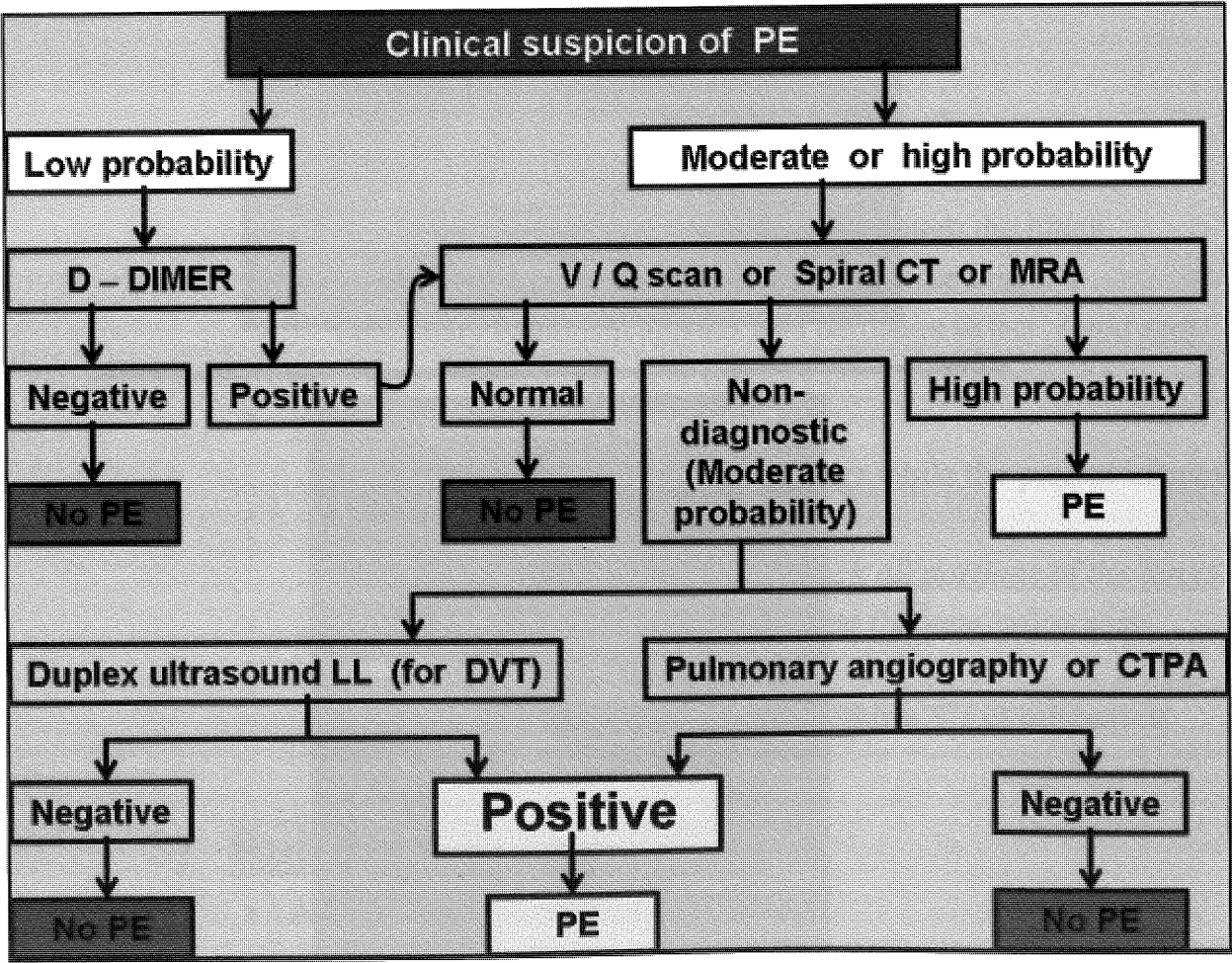
- Highly sensitive.
- It can detect very small emboli (as small as 2 mm).

5) CTPA

- Computed Tomography Pulmonary Angiography.
- Highly sensitive: especially with the recent multidetector CT.

6) MRA

- It is performed following IV injection of gadolinium.



Diagnostic algorithm for pulmonary embolism

TREATMENT

I. Prophylactic:

1. Prophylaxis & treatment: of the predisposing factors.
2. Prophylactic anticoagulation: in predisposed patients.

II. Therapeutic:

1. Hospitalization in ICU.

2. GENERAL TREATMENT

a) Symptomatic treatment:

- Pain: Pethidine 50 mg IM, but avoid morphine.
- Dyspnea: Oxygen inhalation.
- RVF.
- Shock.

b) Treatment of the original source of embolism.

3. SPECIFIC TREATMENT

If the patient is hemodynamically unstable (Shock or HF)

a) Thrombolytic therapy: “Streptokinase”

- 250,000 units IV bolus followed by 100,000 units / hour,
for 24-72 hours, followed by anticoagulation.

b) Pulmonary embolectomy:

- In the patient who does not respond to thrombolytic therapy.
- Presence of a contraindication to thrombolytic therapy.

If the patient is hemodynamically stable

Further thrombosis & embolization are prevented by:

a) Anticoagulation:

- Heparin IV for: **5 days**.
- Heparin & oral anticoagulants, e.g. warfarin for: **5 days**.
- Warfarin for: **3 months** at least.

b) Inferior vena cava interruption:

- Indication: contraindication to anticoagulants.
- Method: Insertion of a filter or an umbrella.

ARRHYTHMIAS

DEFINITION

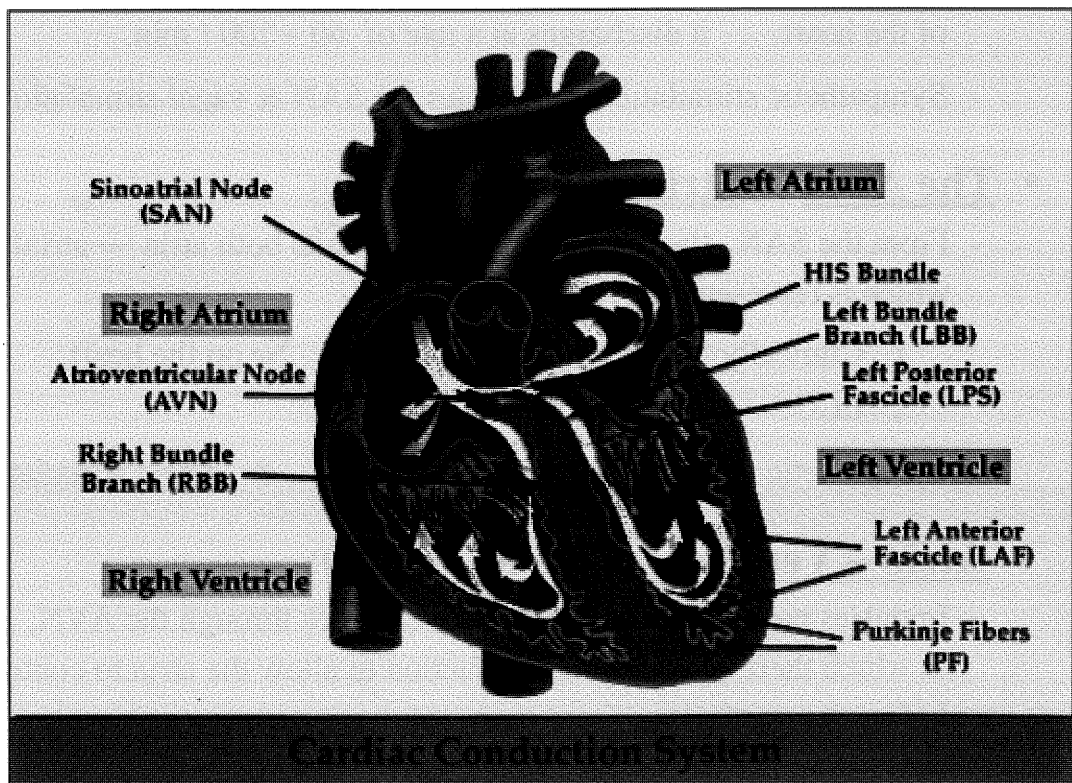
- Arrhythmia is abnormality of the cardiac rhythm or rate.

What happens normally ?? (the normal sequence of cardiac impulse)

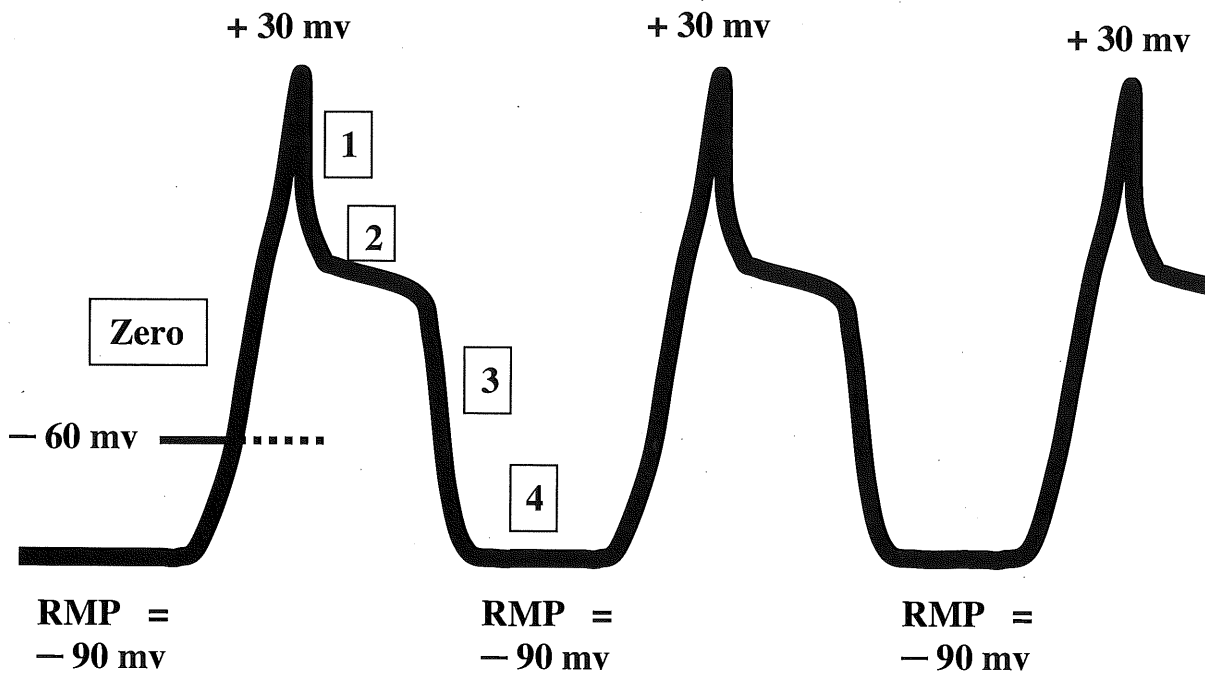
- **Normally:** HR is 60 – 100 beats / min. & the rhythm is regular.
- **Sino – Atrial Node (SAN):**
 - The cardiac impulses arise from the SAN, which is the pace – maker of the heart; it discharges at a rate of 60 – 100 / min.
 - Sympathetic stimulation accelerates the SAN & vagal stimulation slows it.
- **The Atria (atrial activation):**
 - From the SAN, the impulses spread to excite both atria → atrial contraction, which is represented by:
 1. Clinically: a wave in neck veins, S4 on the heart.
 2. ECG: p wave.
 - Sympathetic stimulation ↑ atrial excitability & vagal stimulation ↓ it.
- **Atrio – Ventricular Node (AVN):**
 - Impulses pass from the atria to the ventricles through the AVN, in which there is a delay of conduction to allow the atria to contract before the ventricles.
 - The passage of impulses from the atria to the ventricles is represented by:
 - o ECG: PR interval.
 - Normally, the AVN allows passage of impulses from the atria to the ventricles but not the reverse (no retrograde conduction).
 - Sympathetic stimulation accelerates conduction in the AVN, while vagal stimulation slows it.

• **The Ventricles** **(ventricular activation):**

- Impulses pass from the AVN to the Bundle of His in the inter-ventricular septum, then along the 2 bundle branches & finally Purkinje fibers to terminate in the ventricular myocardium → ventricular contraction which is represented by:
 1. Clinically: radial pulse & first heart sound (S₁).
 2. ECG: QRS complex.
- The ventricles are supplied by sympathetic fibers only (**no vagal supply**).



Action potential of the cardiac cell



Phases of the action potential

- Zero = rapid **D**epolarization (entry of Na^+ inside the cell)
- 1 = early rapid **R**epolarization (exit of K^+ outside the cell)
- 2 = **P**lateau (exit of K^+ is equilibrated by Ca^{++} influx)
- 3 = final rapid **R**epolarization (exit of K^+ outside the cell)
- 4 = **RMP** (**R**esting **M**embrane **P**otential)

What happens abnormally ?? (Mechanisms of arrhythmias)

(A) Disturbance of impulse formation:

1. Disturbed normal automaticity:

- The natural pacemaker cells (SAN) show spontaneous depolarization during diastole (phase 4) of the action potential, resulting in a new action potential when the electric threshold is reached.
- Such diastolic depolarization may be accelerated or slowed by: changing its rate.
- These changes will produce sinus tachycardia or sinus bradycardia, respectively.

2. Abnormal automaticity:

- Normally:
The normal working myocardial cells remain in a resting state at a high negative RMP (-90 mV), depolarizing only when stimulated by SAN.
- In certain diseases: (e.g. ischemia),
RMP of these cells is reduced (-60 mv), Such cells may exhibit spontaneous diastolic depolarization & act as abnormal pace – makers, independent on the SAN; This may be responsible for:
atrial or ventricular or nodal (junctional) arrhythmias.

(B) Disturbance of impulse conduction:

1. Decreased conduction:

- There is loss of the ability of the action potential to stimulate the myocardial cells, e.g. heart block.

2. Re-entry: *

- A wave of depolarization may be forced to travel in one direction around a ring of cardiac tissue.
- A circus movement will result producing a tachycardia, e.g. paroxysmal tachycardias.

* Two conditions are required for re-entry to occur.

INVESTIGATIONS

A) For diagnosis of the TYPE of arrhythmia:

- ★ 1. ECG (resting & ambulatory). ★
- 2. EPS (electrophysiologic study).

B) For diagnosis of the CAUSE of arrhythmia:

- ★ 1. ECG. ★

2. IMAGING:

- CXR.
- Echocardiography
- Cardiac catheterization & angiocardiology.

3. OTHERS:

- Cardiac enzymes: for AMI.
- Thyroid function tests: for Hyperthyroidism.
- Serum K: for ↓ K or ↑ K.

CLINICAL CLASSIFICATION

A) SINUS RHYTHM DISTURBANCES:

1. Sinus tachycardia.
2. Sinus bradycardia.
3. Sinus arrhythmia.

B) PATHOLOGICAL TACHY – ARRHYTHMIAS:

1. Supraventricular tachycardia (SVT).
2. Atrial flutter.
3. Ventricular tachycardia (VT).
4. Atrial fibrillation (AF).

C) PATHOLOGICAL BRADY – ARRHYTHMIAS:

1. Nodal (Junctional) rhythm.
2. Heart block.
3. Sick sinus syndrome.

D) OTHERS:

1. Premature beats (Extrasystoles).
2. Pre-excitation syndromes.

SINUS TACHYCARDIA

DEFINITION

- The SAN discharges at a rapid rate > 100 / minute.

ETIOLOGY

1. Physiological:

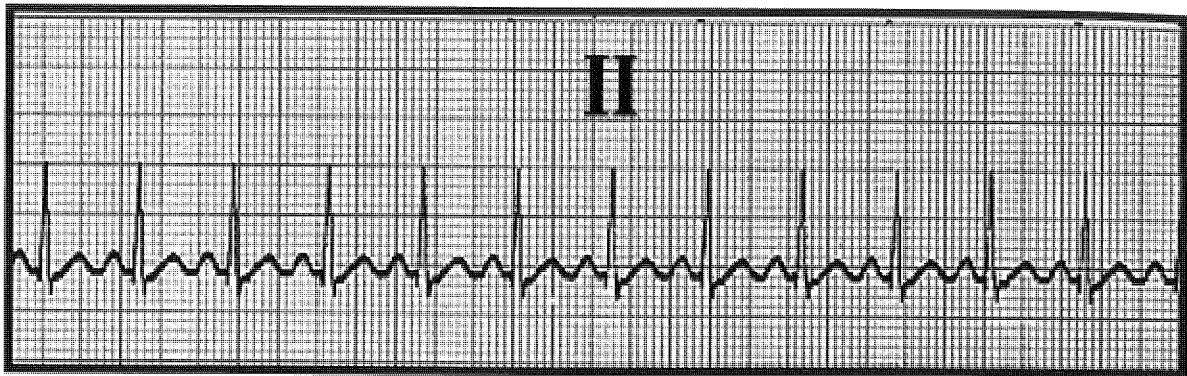
- **E**xercise.
- **E**motions.
- Pregnancy.
- Infancy.

2. Pathological:

- | | |
|----------------------------|-----------------------------------|
| • Fever, | Anemia. |
| • Shock, | Myocarditis. |
| • <u>H</u> yperthyroidism, | <u>H</u> yperdynamic circulation. |
| • <u>H</u> ypovolemia, | <u>H</u> ypotension. |
| • HEART FAILURE. | |
| • PULMONARY EMBOLISM. | |

3. Pharmacological:

- | | |
|--------------------------------|--------------------------|
| • Sympathomimetic drugs: | e.g. <u>A</u> drenaline. |
| • Parasympatholytic drugs: | e.g. <u>A</u> tropine. |
| • CCBs: | e.g. <u>N</u> ifedipine. |
| • Thyroid hormones. | |
| • Nicotine, Caffeine, Alcohol. | |



Sinus tachycardia

TREATMENT

1. TTT of the cause, e.g. antithyroid drugs for hyperthyroidism.
2. Sedatives: may be needed.
3. β -blockers: e.g. propranolol, in severe cases.

SINUS BRADYCARDIA

DEFINITION

- The SAN discharges at a slow rate < 60 / minute.

ETIOLOGY

1. Physiological:

- Athletes.
- Asleep.

2. Pathological:

- Hypothermia.
- Hypothyroidism.
- Increased vagal stimulation, e.g. Vasovagal syncope.
- Increased intracranial tension, e.g. Brain tumours.
- CHOLESTASIS: Obstructive jaundice.

3. Pharmacological:

- Parasympathomimetic drugs: e.g. Neostigmine.
- Sympatholytic drugs: e.g. β -blockers.
- CCBs: e.g. Verapamil & diltiazem.
- DIGITALIS.

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. PALPITATION:

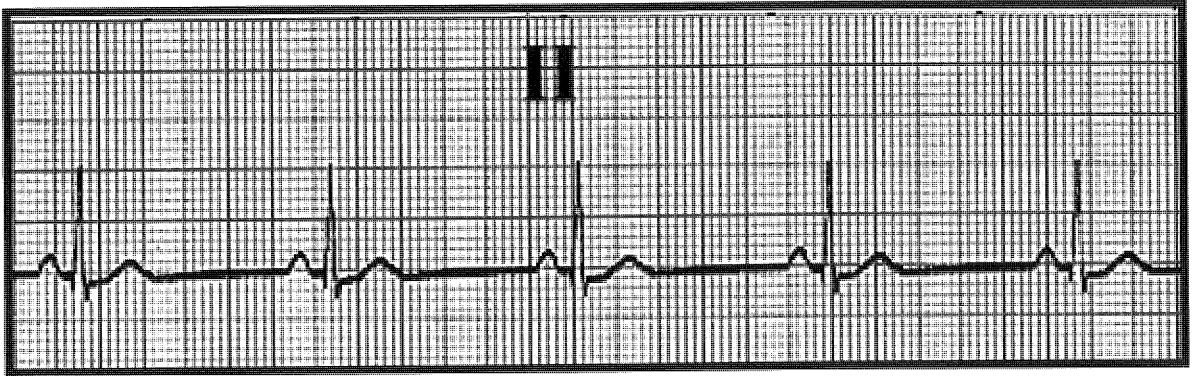
<u>slow</u> ,	<u>regular</u> .
<u>gradual</u> onset,	<u>gradual</u> offset.
2. Symptoms of: low cardiac output.
3. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:
 - Rhythm: regular.
 - Rate: < 60 / minute (50 – 60 / minute, or less).
 - Response to exercise:
 - *Gradual acceleration:* of the rate.
 - *Gradual slowing:* to the original rate on stopping exercise.
2. NECK VEINS:
 - Normal slow waves with the same rate of pulse.
3. AUSCULTATION:
 - Normal or weak S₁.

ECG

- QRS:
 - Rhythm: regular.
 - Rate: 50 – 60 / min, (or less).
 - Duration: normal.
- P wave:
 - Normal.



Sinus bradycardia

TREATMENT

1. TTT of the cause, e.g. L – thyroxin for hypothyroidism.
2. Atropine: may be needed.

SUPRAVENTRICULAR TACHYCARDIA

DEFINITION

- It is an tachy – arrhythmia originating from above the ventricle.

TYPES

1. *Atrial tachycardia*: the arrhythmia originates from the Atria.
2. *Nodal tachycardia*: the arrhythmia originates from the AVN.

ETIOLOGY

- It usually occurs in a normal heart, & therefore its presence does not always denote an ORGANIC HEART DISEASE.

1. Physiological: occurs in a normal heart (no organic heart disease).

2. Pathological: (organic heart disease)

- CAD (especially AMI).
- Cardiomyopathy (and myocarditis).
- *Congenital heart disease*.
- *RHD*.
- Hypertension.
- Hyperthyroidism.
- PRE – EXCITATION SUNDROMES: e.g. WPW syndrome.

3. Pharmacological:

- DIGITALIS.
- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. PALPITATION:

rapid, regular.
sudden onset, sudden offset.

2. Symptoms of: low cardiac output.

3. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:

- Rhythm: regular.
- Rate: 120 – 250 / minute.
- Response to carotid massage:
 - *Sudden disappearance: of the arrhythmia may occur.*

2. NECK VEINS:

- In atrial tachycardia: normal rapid waves with the same rate of pulse.
- In nodal tachycardia: regular cannon waves with the same rate of pulse.

3. AUSCULTATION:

- In atrial tachycardia: accentuated S₁.
- In nodal tachycardia: regular cannon sounds.

ECG

• QRS:

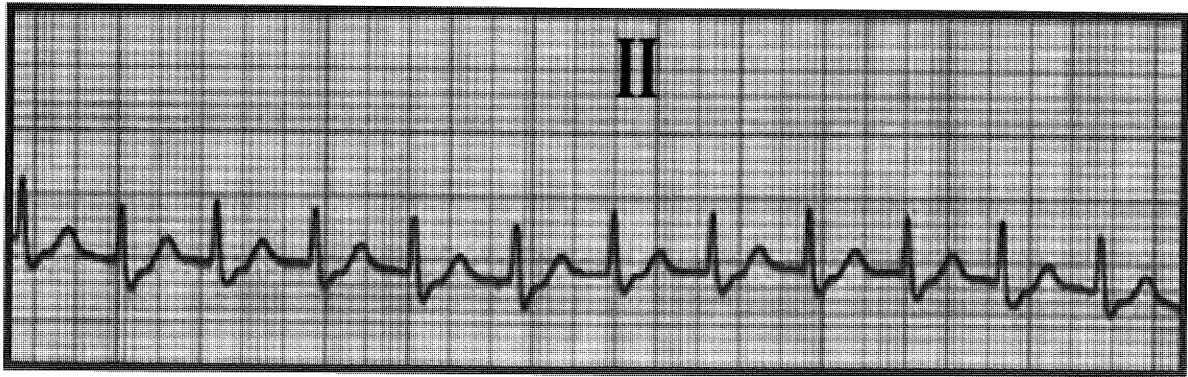
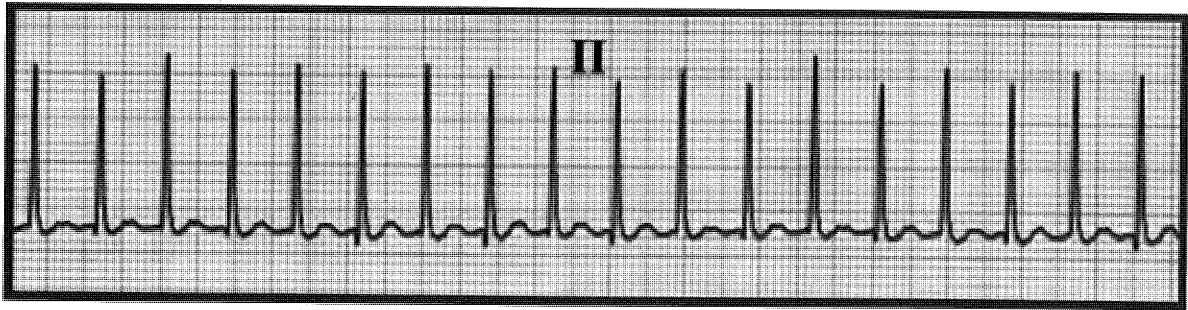
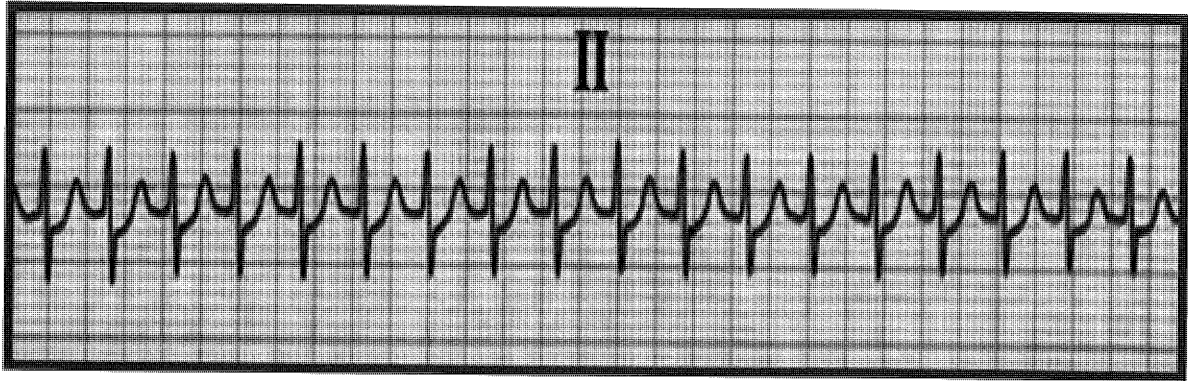
- Rhythm: regular.
- Rate: 120 – 250 / min.
- Duration: normal.

• P wave:

- In atrial tachycardia: deformed.
- In nodal tachycardia: absent or inverted.

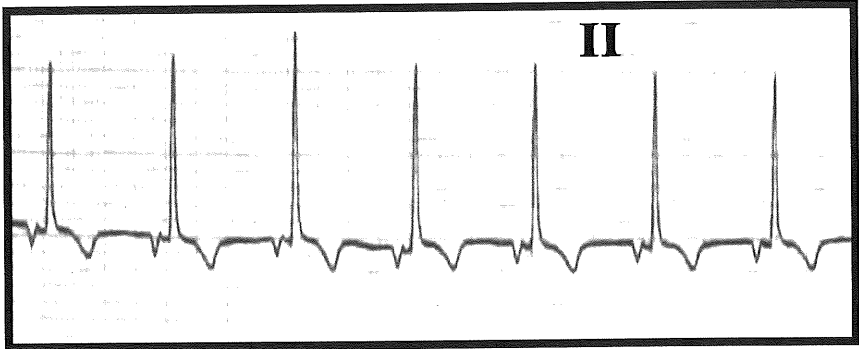
Nodal tachycardia

“Absent p”

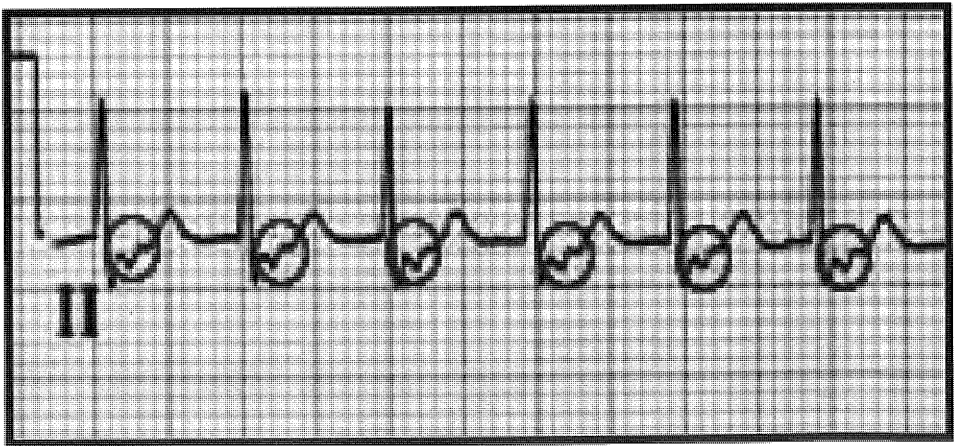


Nodal tachycardia

“Inverted p”



“Before QRS”



“After QRS”

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

1. Vagal stimulation: carotid sinus massage.
2. Slow the ventricular rate:
 - **A**denosine: 6 mg IV.
 - **β**-blocker (propranolol): 5 mg IV.
 - **C**CB (verapamil): 5 mg IV.
 - **D**igitalis: 1 mg IV.
3. Restore sinus rhythm (Cardioversion): (after ↓ the ventricular rate)
 - Chemical cardioversion: Class IA, IC or class III drugs.
 - Electrical cardioversion (DC): if chemical cardioversion fails.

B. If the patient is hemodynamically unstable:

1. DC cardioversion.
2. Overdrive pacing:
 The atria are paced at a faster rate than the tachycardia rate.
 Sudden cessation of pacing is usually followed by:
 “restoration of sinus rhythm”.

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class IA, IC or class III anti – arrhythmic drugs.

B. Intervention:

- Ablation of focus: Catheter (Radiofrequency energy) or surgery.

C. TTT of the cause.

ATRIAL FLUTTER

DEFINITION

- A tachycardia in which the atria discharge at a regular rapid rate: 240 – 440 / min.
- A physiological block occurs in the AVN (2:1, or 3:1, or 4:1 block).
- Therefore only $\frac{1}{2}$, or $\frac{1}{3}$, or $\frac{1}{4}$ of the atrial impulses will pass to the ventricles.
- The average ventricular rate will be: $\frac{1}{2}$, or $\frac{1}{3}$, or $\frac{1}{4}$ the atrial rate.
- The block may be:
 - Fixed (e.g. 2:1), or,
 - Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).

TYPES

1. *Type I (common, typical):* the atrial rate is 240 – 340 / min. $\approx$ 300 / min.
2. *Type II (rare):* the atrial rate is 340 – 440 / min. $\approx$ 400 / min

ETIOLOGY

- It usually occurs in a patient with ORGANIC HEART DISEASE, However,
It may occur in a normal heart.

1. Physiological: *may* occur in a normal heart (no organic heart disease).
2. Pathological: (organic heart disease)
 - CAD (especially AMI).
 - Cardiomyopathy (and myocarditis).
 - *Congenital heart disease.*
 - *RHD.*
 - Hypertension.
 - Hyperthyroidism.
 - PRE – EXCITATION SYNDROMES: e.g. WPW syndrome.
3. Pharmacological:
 - DIGITALIS.
 - Sympathomimetic drugs.
 - Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. PALPITATION:

rapid, regular (or irregular).
sudden onset, sudden offset (or → AF).

2. Symptoms of: low cardiac output.

3. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:

- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **150** or 100 or 75 / min. (according to AV block).
- Response to carotid massage: (↑ AV block from 2:1 → 3:1 → 4:1)
 - *Stepwise slowing:* of the rate (150 → 100 → 75 / min).
 - *Stepwise acceleration:* to the original rate on stopping massage.

2. NECK VEINS:

- Multiple “a” waves: **double** or triple or quadruple the rate of pulse.

3. AUSCULTATION:

- Accentuated S₁.

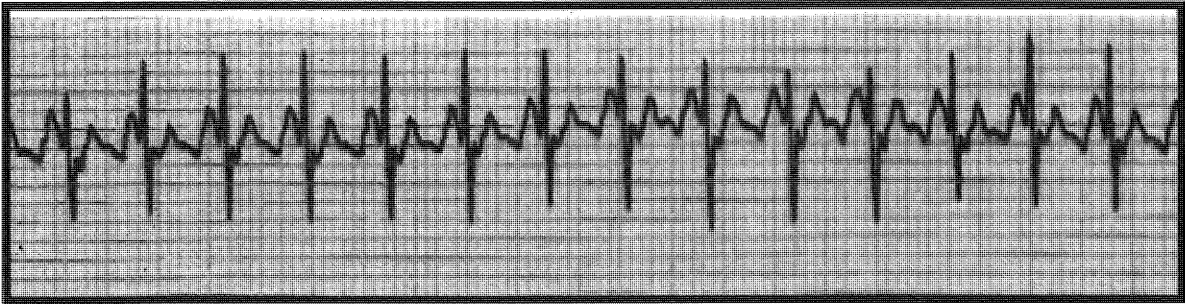
ECG

• QRS:

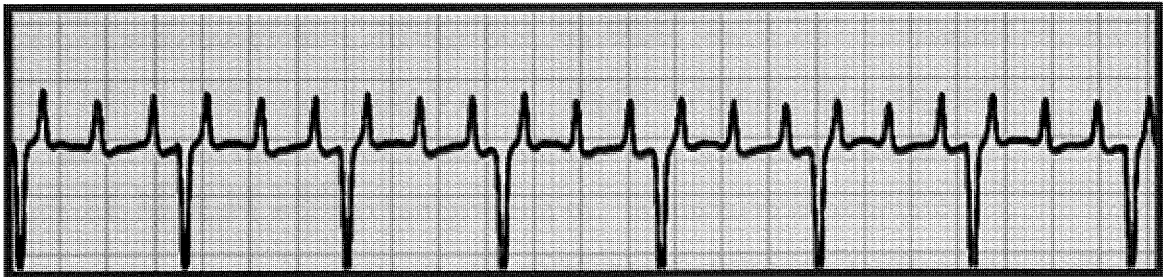
- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **150** or 100 or 75 / min. (according to AV block).
- Duration: normal.

• P wave:

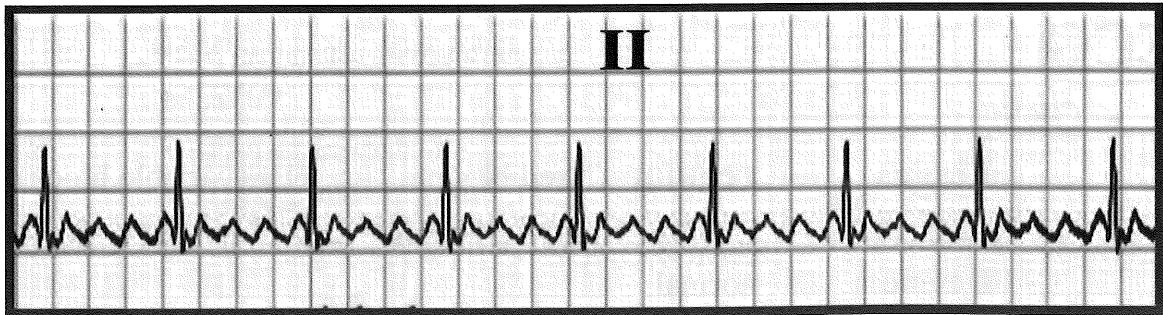
- Replaced by: multiple “Flutter waves” at a rate of 240 – 340 / min.
- Typically: **“Saw – tooth appearance”**.



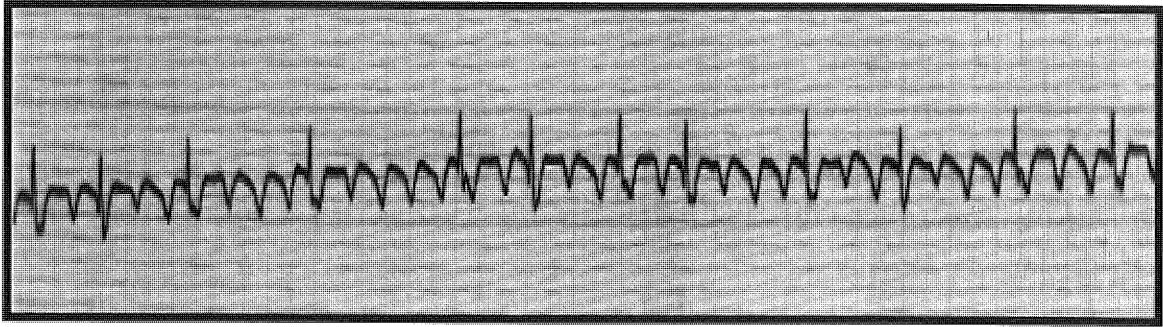
Regular “Fixed block 2 : 1”



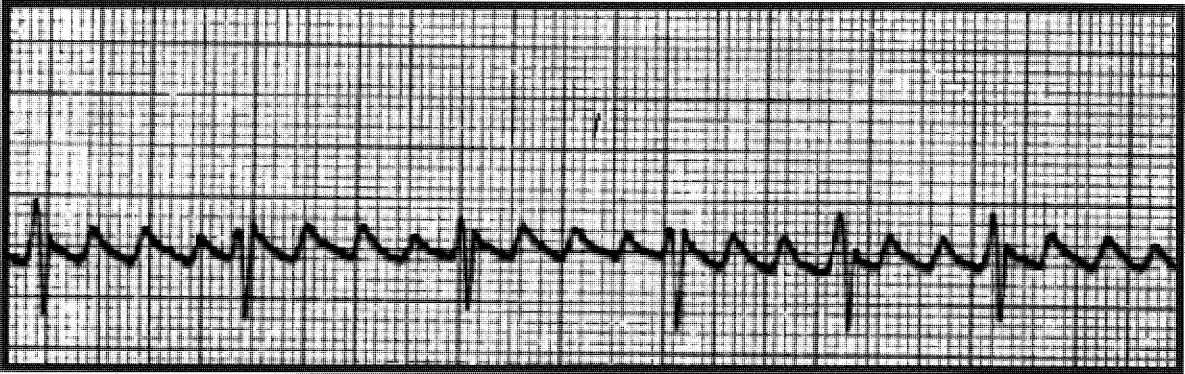
Regular “Fixed block 3 : 1”



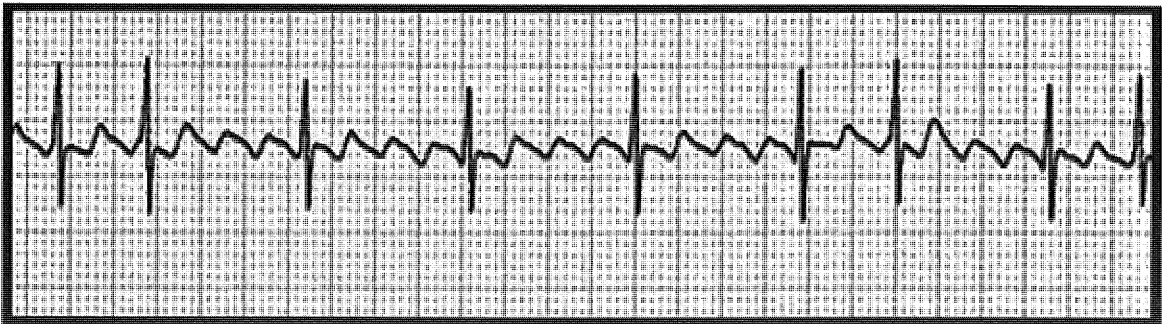
Regular “Fixed block 4: 1”



Irregular “Variable block”



Irregular “Variable block”



Irregular “Variable block”

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

1. Slow the ventricular rate:

- **A**denosine: 6 mg IV.
- **β**-blocker (propranolol): 5 mg IV.
- **C**CB (verapamil): 5 mg IV.
- **D**igitalis: 1 mg IV.

Digitalis, by increasing atrial automaticity, may convert Atrial flutter into AF. If the drug is then stopped, sinus rhythm may be restored.

2. Restore sinus rhythm (Cardioversion): (after ↓ the ventricular rate)

- Chemical cardioversion: Class IA, IC or class III drugs.
- Electrical cardioversion (DC): if chemical cardioversion fails.

B. If the patient is hemodynamically unstable:

1. DC cardioversion.

2. Overdrive pacing:

The atria are paced at a faster rate than the tachycardia rate.
Sudden cessation of pacing is usually followed by:
“restoration of sinus rhythm”.

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class IA, IC or class III anti – arrhythmic drugs.

B. Intervention:

- Ablation of focus: **Catheter** (Radiofrequency energy) or **surgery**.

C. TTT of the cause.

VENTRICULAR TACHYCARDIA

DEFINITION

- It is an arrhythmia originating from the VENTRICLE that presents with:
 - Three or more successive ventricular premature beats.
 - Rapid Regular tachycardia at a rate of: 120 – 250 / min.
- Since there is no retrograde conduction in the AVN, there will be AV dissociation:
 - The ventricles will be controlled by the ventricular focus: (VR 120 – 250 / min).
 - The atria will be controlled by the SAN: (AR: 60 – 100 / min).

TYPES

1. *Sustained*: persists more than 30 sec. or causes hemodynamic instability.
2. *Non-sustained*: persists less than 30 sec. with no hemodynamic instability.

ETIOLOGY

- It *vvv rarely* occurs in a normal heart, & therefore its presence almost always denotes an ORGANIC HEART DISEASE.

1. Pathological:

- CAD (especially AMI).
- Cardiomyopathy (and myocarditis).
- *Congenital heart disease*.
- *RHD*.
- Hypertension.
- Hyperthyroidism.
- PRE – EXCITATION SUNDROMES: e.g. WPW syndrome.

2. Pharmacological:

- DIGITALIS.
- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. Palpitation: rapid, regular.
 sudden onset, sudden offset.
2. Symptoms of: low cardiac output & shock are more common.
3. Precipitation of: angina & heart failure in susceptible patients.
4. SUDDEN DEATH: if converted to Ventricular fibrillation (VF).

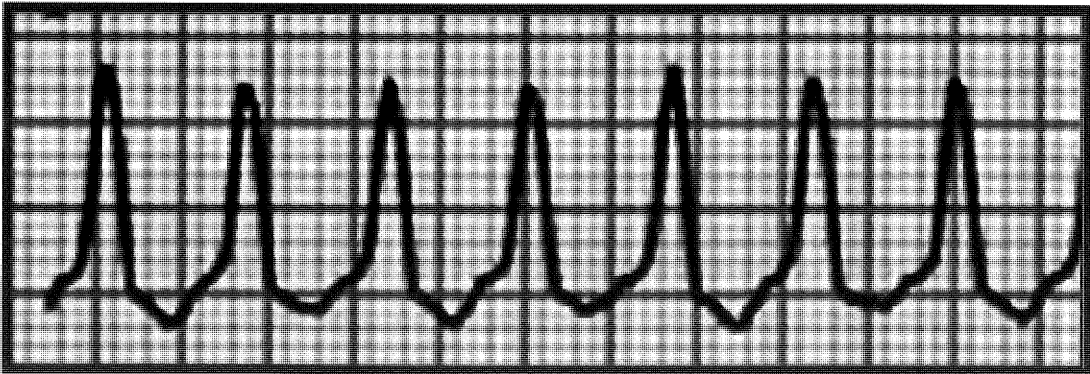
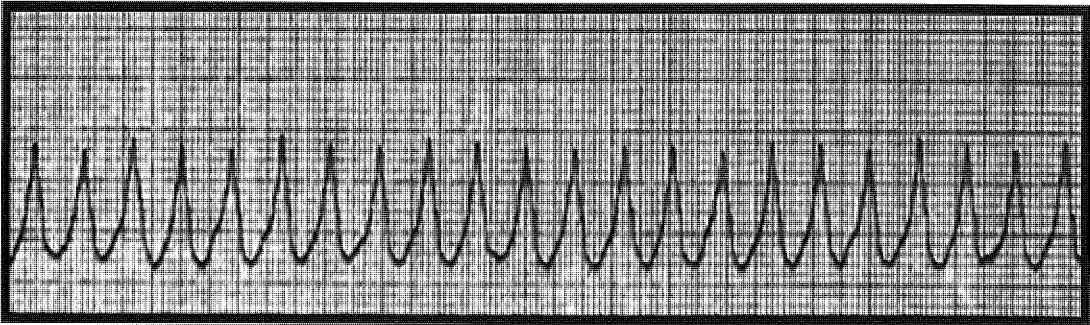
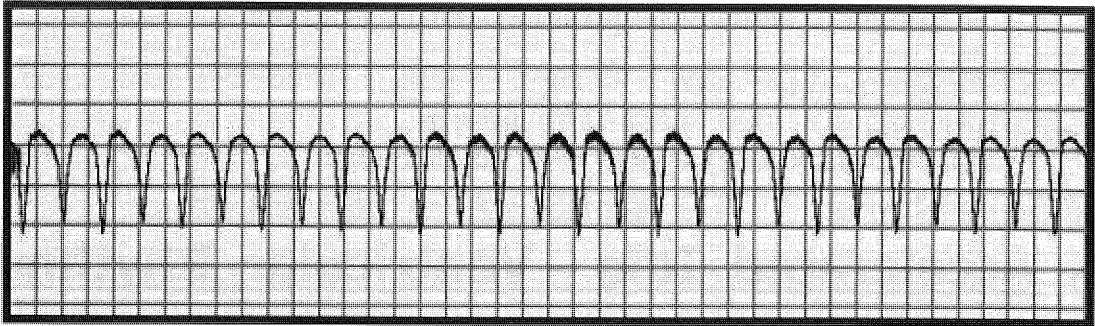
SIGNS

1. PULSE:
 - Rhythm: regular
 - Rate: 120 – 250 / min.
 - Response to carotid massage: no response (no vagal supply to ventricles).
2. NECK VEINS:
 - “a” waves: normal rate (60 – 100 / min) & less than the pulse rate.
 - OCCASIONAL CANNON WAVES.
3. AUSCULTATION:
 - OCCASIONAL CANNON SOUNDS.
 - Variable intensity of S₁.

ECG

- QRS:
 - Rhythm: regular.
 - Rate: 120 – 250 / min.
 - Duration: wide.
- P wave: (may not appear)
 - Normal in rate (60 – 100 / minute) & shape.
 - Comes before, after or is hidden by the QRS (AV dissociation).

Ventricular tachycardia



SPECIAL TYPES OF VT

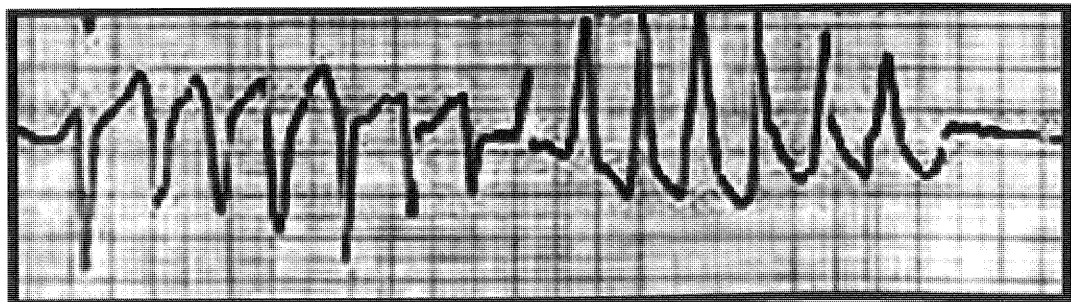
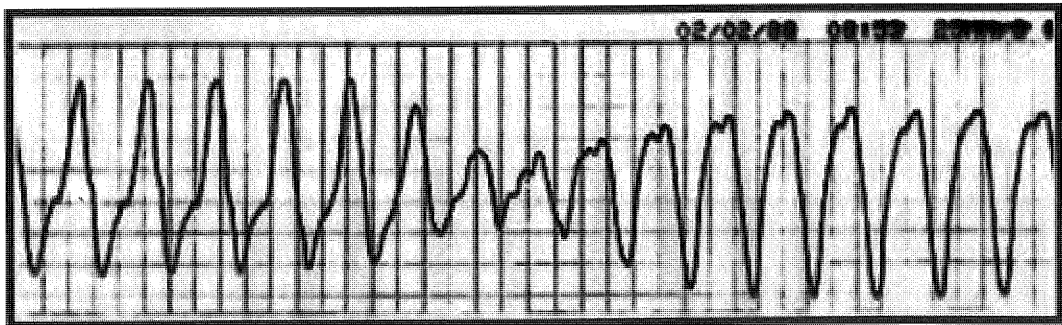
1. Torsade de pointes:

- QRS complexes change continuously & rapidly & irregularly from an upright to an inverted position (twisting of points).
- Causes include: AMI, $\downarrow$ K, $\downarrow$ Ca.
- It is serious & may cause VF & sudden death.

2. Accelerated Idio – Ventricular Rhythm:

- An ectopic ventricular pacemaker discharges at a rate: 60 – 120 / min, and controls the ventricles only resulting in a slow VT.
- Causes include: AMI, post-coronary thrombolysis (*reperfusion arrhythmias*).
- It is transient & rarely causes hemodynamic disturbances.

Torsade de pointes



ATRIAL FIBRILLATION

DEFINITION

- A form of tachycardia in which the atria discharge at a rate: 400 – 600 / min.
- An irregular block occurs in the AVN allowing only some impulses to pass to the ventricles in an IRREGULAR manner.
- Therefore, the ventricular beats will be:
 - Rhythm: markedly irregular.
 - Rate: 100 – 160 / min.
 - Force: “variable”.

ETIOLOGY

1. Primary (Idiopathic): “LONE AF” (not secondary to any disease).
 - It is more common in old age, and may be slow.
2. Pathological: (Secondary to different diseases)
 - CAD (especially AMI).
 - Cardiomyopathy (and myocarditis).
 - Chest disease, especially COPD.
 - Constrictive pericarditis.
 - *Congenital heart disease*, especially ASD.
 - *RHD*, especially MS.
 - Hypertension.
 - Hyperthyroidism.
 - PRE – EXCITATION SUNDROMES: e.g. WPW syndrome.
3. Pharmacological:
 - DIGITALIS.
 - Sympathomimetic drugs.
 - Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. PALPITATION:

rapid, irregular.
sudden onset, sudden offset.

2. Symptoms of: low cardiac output.
3. Precipitation of: angina & HF in susceptible patients.
4. COMPLICATIONS: atrial thrombosis & embolization.

SIGNS

1. PULSE:

- Rhythm:
 - Marked **irregularity** (in rhythm & volume).
 - Pulse deficit: > 10 beats / minute.
- Rate: 100 – 160 / min.

Causes of SLOW AF

1. Drugs: Digitalis or β -blocker.
2. Lone AF.
3. Associated heart block.

- Response to carotid massage:
 - *Gradual slowing*: due to ↓ AV conduction.
- Response to exercise:
 - ↑ *irregularity*: due to ↑ AV conduction.

2. NECK VEINS:

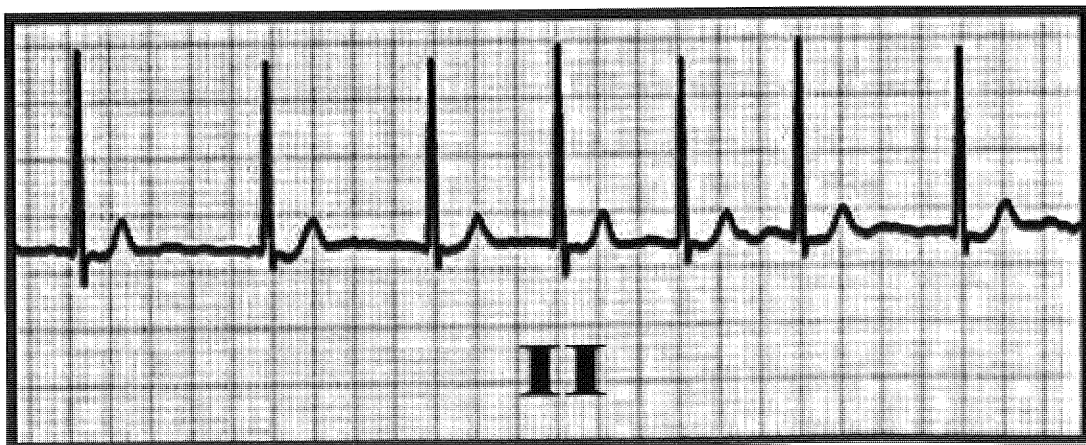
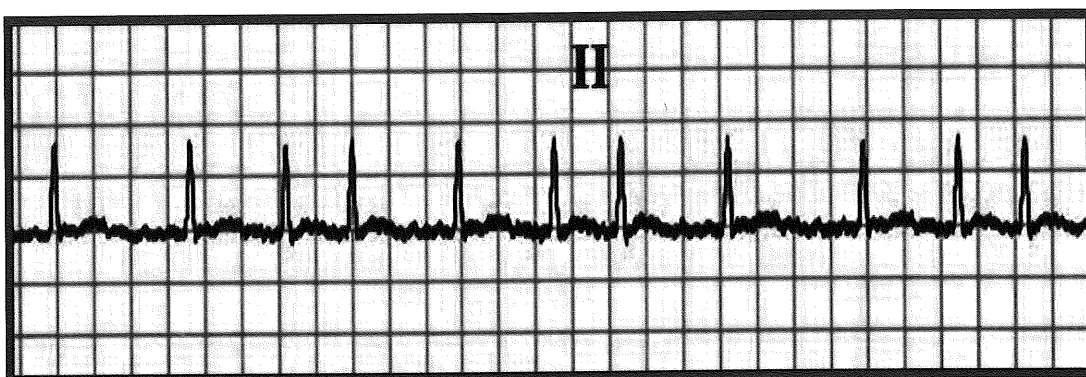
- “a” waves: **absent**.
- Systolic expansion.

3. AUSCULTATION:

- Variable intensity of S₁.

ECG

- QRS:
 - Rhythm: markedly irregular.
 - Rate: 100 – 160 / min.
 - Duration: normal.
- P wave: **“Absent p”**
 - Replaced by: fibrillation waves (irregular vibrations).



TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

1. Restore sinus rhythm:

“CARDIOVERSION”

- INDICATIONS:

- Recent onset AF: < 12 months duration.
- Absence of: embolizaion or history of embolization.
- Absence of: atrial thrombus **or** significant AE.

- CONTRA – INDICATIONS:

- Longstanding AF: > 12 months duration.
- Presence of: embolizaion or history of embolization.
- Presence of: atrial thrombus **or** significant AE.

- METHODS:

- Chemical cardioversion: Class IA, IC or class III drugs.
- Electrical cardioversion (DC): if chemical cardioversion fails.

- PRECAUTIONS:

- Anticoagulation should be given:
 - i. Before cardioversion (3 – 4 weeks).
 - ii. After cardioversion (3 – 4 weeks).
- Digitalis should be stopped before **electrical** cardioversion (DC).

2. Slow the ventricular rate:

“RATE CONTROL”

- Indication: when cardioversion fails or is contraindicated.
- Drugs: **β**-blocker (propranolol), **C**CB (verapamil), **D**igitalis.

B. If the patient is hemodynamically unstable:

1. DC cardioversion.

2. Overdrive pacing:

The atria are paced at a faster rate than the tachycardia rate.

Sudden cessation of pacing is usually followed by:

“restoration of sinus rhythm”.

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class IA, IC or class III anti – arrhythmic drugs.

B. Intervention:

- Ablation of focus: **Catheter** (Radiofrequency energy) or **surgery**.

C. TTT of the cause.

Indications of Anticoagulation in AF

1. Cardioversion (Before & after).
2. Embolization or presence of atrial thrombus. (INR: 2.5 – 3.5)
3. Valvular AF, especially MS. (INR: 2 – 3)
4. Non – valvular AF with associated risk factors for thromboembolism:
 - Age > 75 years.
 - Presence of: CAD, HF, HTN.
 - Associated DM.

} (INR: 2 – 3)

PREMATURE BEATS (EXTRASYSTOLES)

DEFINITION

- They are **ectopic** cardiac impulses occurring before the expected sinus impulse causing premature beats.
- When the normal sinus impulse arises, the heart will not respond because it will be in the: "**Refractory period**".
- These **ectopic** cardiac impulses may arise:
 1. Supraventricular (from the **Atria** or **AVN**) **or**,
 2. Ventricular (from the **Ventricles**).

ETIOLOGY

1. **Physiological**: *may* occur in a normal heart (no organic heart disease).
 - **E**motions. ★
 - **E**xcessive: *smoking, coffee, tea.*
2. **Pathological**: (organic heart disease)
 - CAD (especially **AMI**).
 - Cardiomyopathy (and myocarditis).
 - *Congenital heart disease.*
 - *RHD.*
 - **H**ypertension.
 - **H**yperthyroidism.
 - HYPOKALEMIA.
1. **Pharmacological**:
 - DIGITALIS.
 - Sympathomimetic drugs.
 - Some anti-arrhythmic drugs e.g., **class I C**.

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. PALPITATION: irregular.

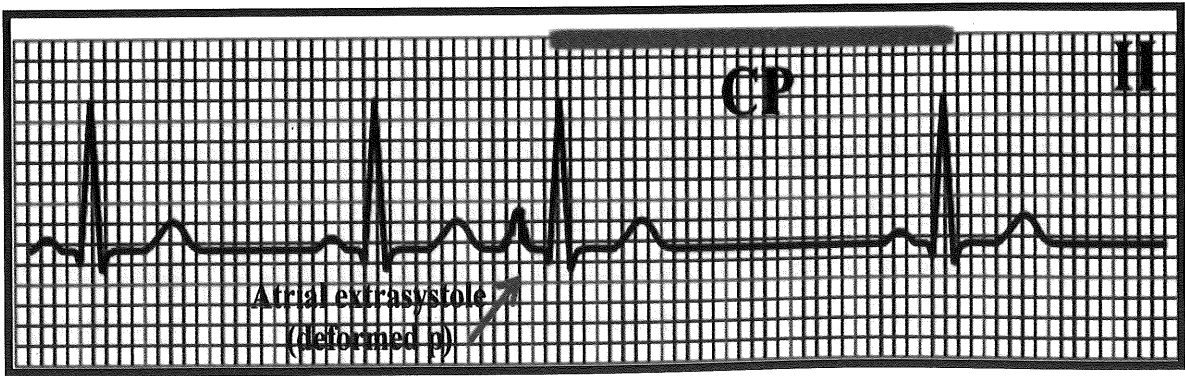
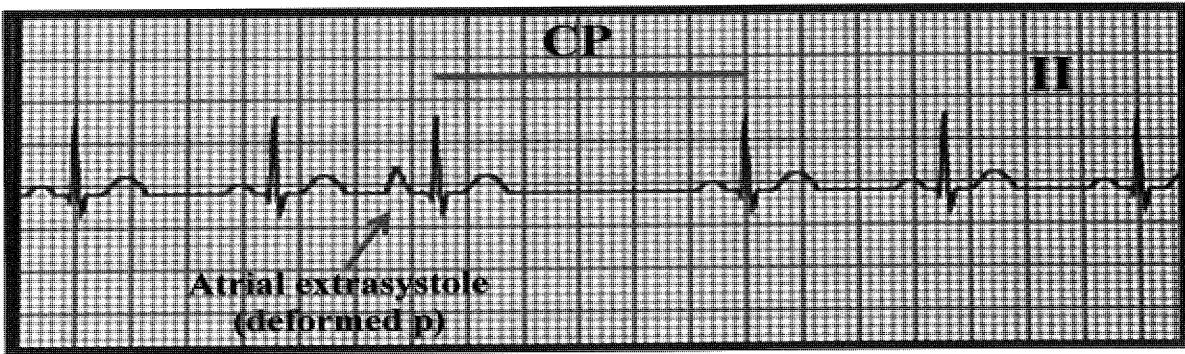
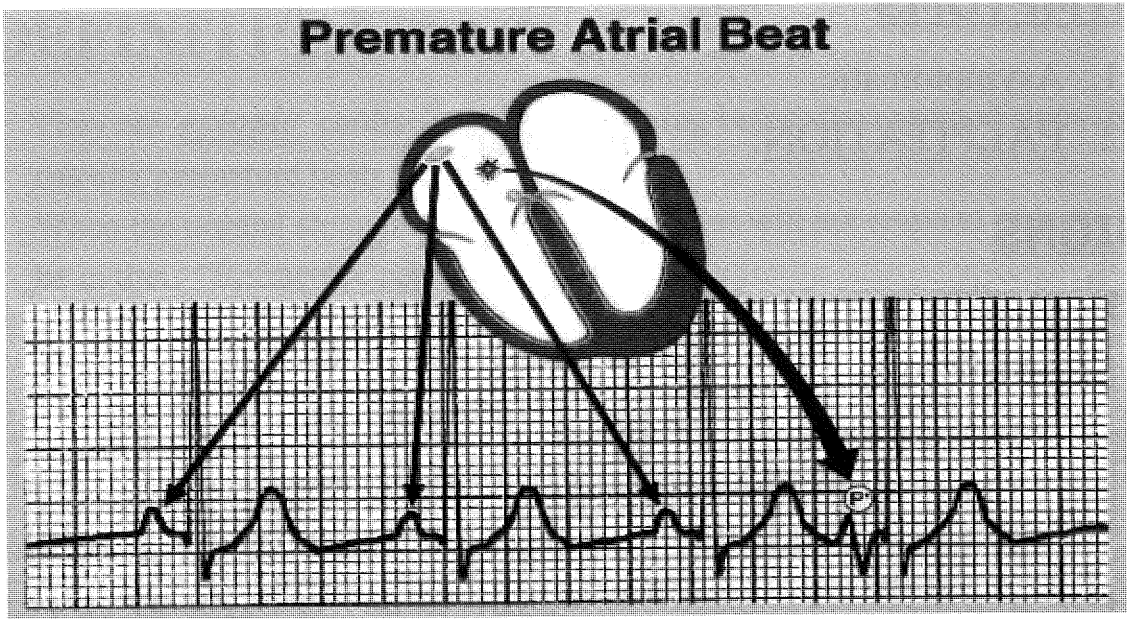
SIGNS

1. PULSE:
 - Rhythm:
 - Occasional **irregularity**.
 - Pulse deficit: < 10 beats / minute.
 - Rate: VARIABLE according to the sinus rhythm (↑, ↓, or normal).
 - Response to exercise: “VARIABLE”
 - ↓ irregularity: due to shortened diastolic period.
 - ↑ irregularity: due to sympathetic stimulation.
2. NECK VEINS:
 - Normal waves with: Occasional **irregularity**.
3. AUSCULTATION:
 - Normal sounds with: Occasional **irregularity**.

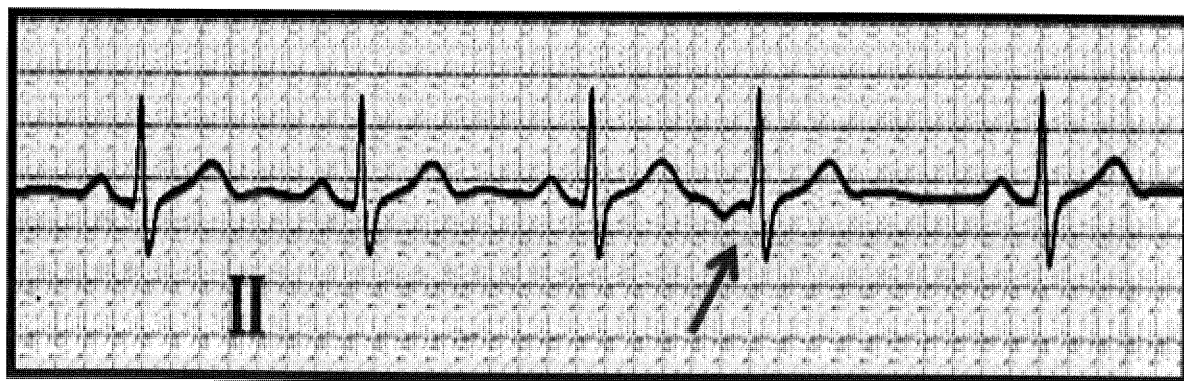
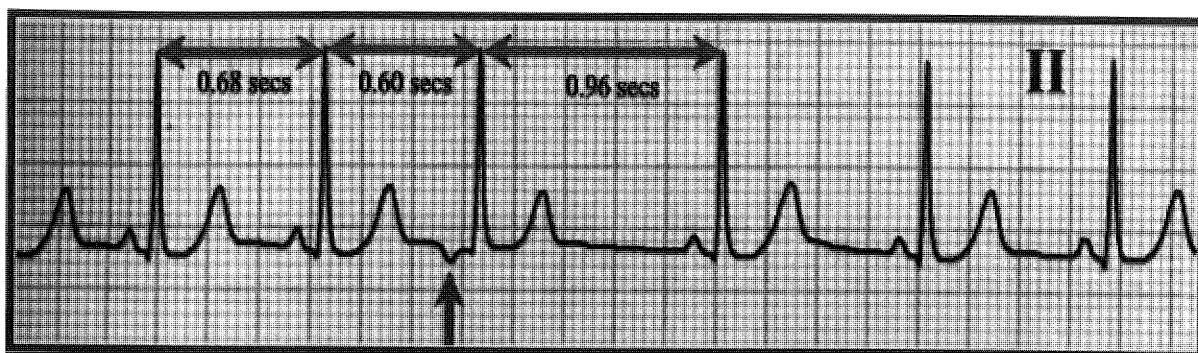
ECG

- Rhythm: irregular (Occasional **irregularity**), but: sinus.
- The premature beat comes early & is followed by a compensatory pause:
 1. Supraventricular (from the Atria or AVN):
 - Atrial beats: deformed p – wave followed by a normal QRS.
 - Nodal beats: absent or inverted p – wave, a normal QRS.
 2. Ventricular (from the Ventricles):
 - Ventricular beats: absent p – wave, wide QRS.

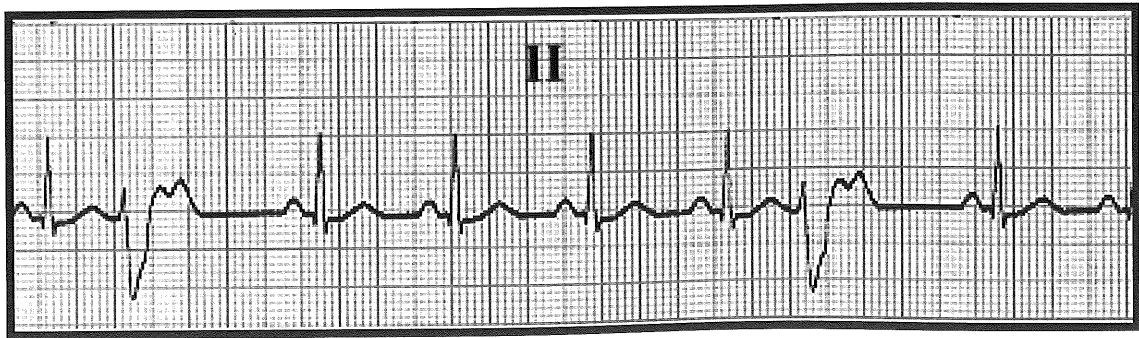
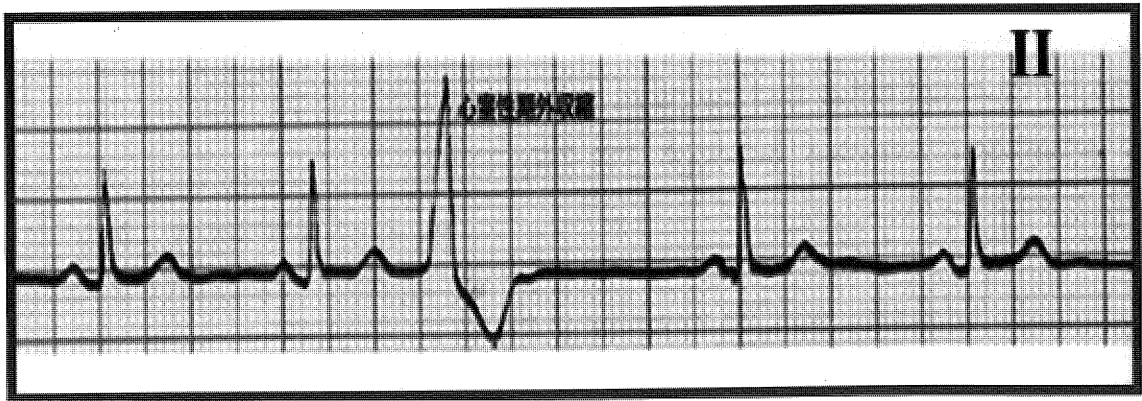
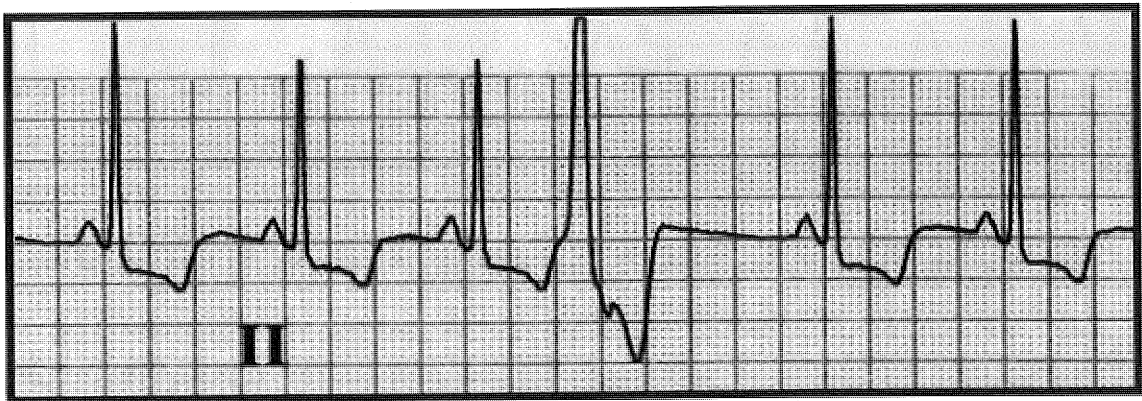
Atrial extrasystole



Nodal extrasystole



Ventricular extrasystole



TREATMENT

1 TTT of the cause.

2 Sedatives: *may be needed.*

3 TTT of extrasystoles:

A) Supraventricular beats:

- They usually need no ttt (asymptomatic).
- They may need β -blockers (Symptomatic: causing palpitation).

B) Ventricular beats:

- They usually need no ttt (asymptomatic).
- They may need Anti – arrhythmic drugs if they are:
 - o Symptomatic: causing palpitation.
 - o Multiple.
 - o Multifocal.
 - o Falling on the preceding T-wave (R on T phenomenon).
 - o Occuring in association with: AMI.
- Anti – arrhythmic drugs are:
 - o In emergency conditions: *e.g. AMI, Digitalis toxicity,*
 - IV Lidocaine is the drug of choice.
 - o In stable conditions:
 - Oral therapy with class I , II , or III drugs.

NODAL (JUNCTIONAL) RHYTHM

DEFINITION

- An abnormal heart rhythm, where the AVN initiates electrical activity of the heart.
- The impulses spread up & down to activate the atria & the ventricles simultaneously.
- There are 2 possibilities:

1. Nodal tachycardia: **“Discussed before”**

- Abnormal automaticity in the AVN overtakes the normal SAN.

2. Nodal rhythm: **“Discussed below”**

- An escape rhythm in which the AVN becomes the pace – maker of the heart discharging at a rate of 40 – 60 / minute, in cases of:
 - o Severe bradycardia: when the SAN discharges at a rate slower than the intrinsic AVN pacemaker.
 - o Heart block: conduction problem between the SAN & the AVN.

ETIOLOGY

- It usually occurs in a patient with ORGANIC HEART DISEASE, However, It may occur in a normal heart.

1. Physiological: *may* occur in a normal heart (↑↑ vagal tone during sleep).

2. Pathological: (organic heart disease)

- CAD (especially **AMI, inferior**).
- Cardiomyopathy (and myocarditis).
- SICK SINUS SYNDROME.

3. Pharmacological:

- DIGITALIS.
- β – blockers.

CLINICAL PICTURE

SYMPTOMS

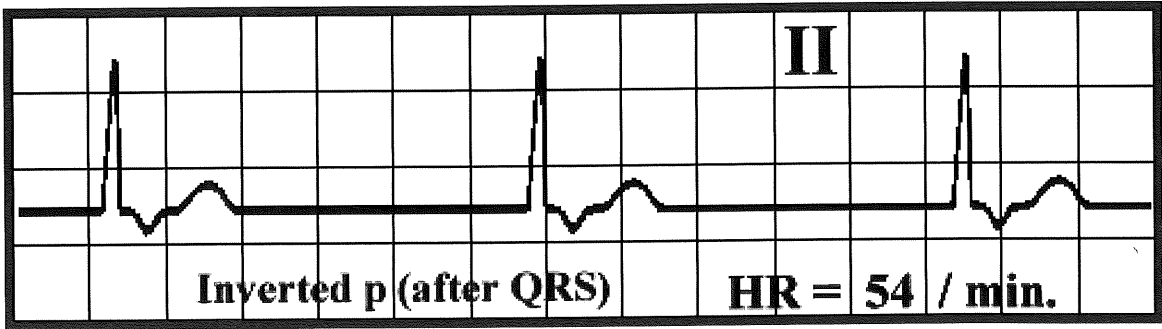
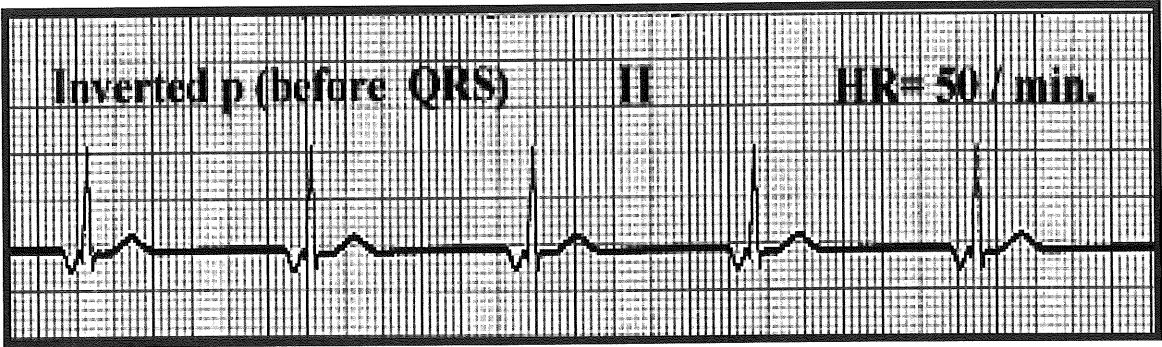
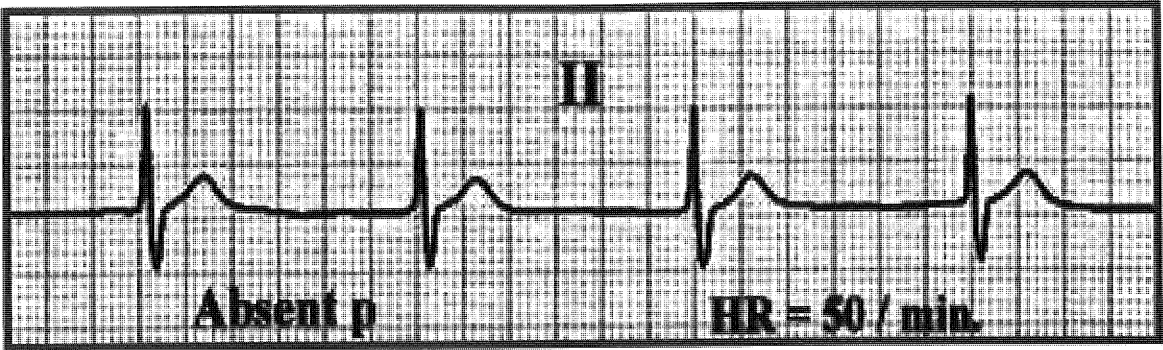
1. Asymptomatic.
2. PALPITATION: regular.
3. Symptoms of: low cardiac output.
4. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:
 - Rhythm: regular.
 - Rate: 40 – 60 / minute.
 - Response to exercise:
 - Reversion to sinus rhythm may occur in some cases.
2. NECK VEINS:
 - Regular cannon waves with the same rate of pulse.
3. AUSCULTATION:
 - Regular cannon sounds.

ECG

- QRS:
 - Rhythm: regular.
 - Rate: 40 – 60 / min.
 - Duration: normal.
- P wave:
 - Absent or inverted.



TREATMENT

1. TTT of the cause, e.g. permanent pacemaker for *sick sinus syndrome*.
2. Atropine: in symptomatic cases.

HEART BLOCK

DEFINITION

- A disease in the *“Electrical system of the heart”*:
- Impairment of impulse conduction at any of the following sites:
 1. SAN (Sinoatrial block): between the SAN & the atria.
 2. AVN (AV block): between the atria & the ventricles.
 3. Bundle branch (BBB): along the bundle branches.

AV BLOCK

ETIOLOGY

1. Pathological: (organic heart disease)
 - CAD (especially AMI, inferior).
 - Cardiomyopathy (and myocarditis).
 - Congenital heart disease.
 - Calcific aortic stenosis.
 - Conduction system fibrosis.
 - HYPERKALEMIA.
2. Pharmacological:
 - **β** – blockers.
 - **CCBs**.
 - **DIGITALIS**.

TYPES = GRADES = DEGREES

- There are 3 degrees of AV block:
 1. First degree: **delayed** conduction of ALL impulses.
 2. Second degree: **no** conduction of Some impulses.
 3. Third degree: **no** conduction of ALL impulses.

First degree

DEFINITION

- Delayed conduction of ALL impulses from the atria to the ventricles.
- ALL p waves are followed by a QRS complex.

CLINICAL PICTURE

SYMPTOMS

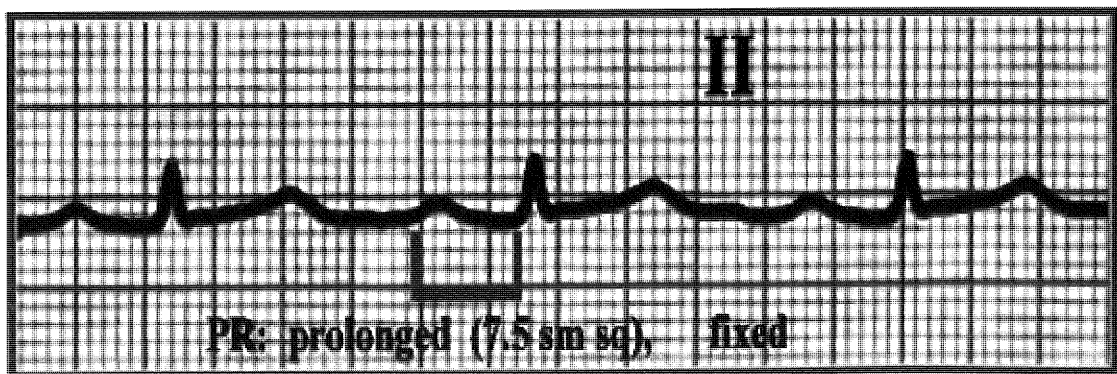
1. Asymptomatic.
2. Symptoms of: *the cause.*

SIGNS

- No clinical signs.

ECG

- PR: *prolonged* > 0.2 sec. (> 5 small squares), *fixed*.



TREATMENT

- TTT of: *the cause.*

Second degree (Incomplete HB)

DEFINITION

- No conduction of Some impulses from the atria to the ventricles.
- Some p waves are not followed by a QRS complex.
- There are 2 types:
 1. Mobitz Type I: (Wenckebach phenomenon)¹ (**Block is in AVN**)
 - Progressive prolongation of the AV conduction time until conduction fails completely & an atrial impulse is blocked.
 2. Mobitz Type II: (**Block is below AVN: Infra-Hisian**)
 - Every 2, 3 or 4 atrial impulses, the AVN transmits only one impulse to the ventricles, (so the block will be: 2:1 or 3:1 or 4:1).
 - The block may be:
 - o Fixed (e.g. 2:1), or,
 - o Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. Symptoms of: the cause.
3. PLUS: In Mobitz Type II,
 - PALPITATION:
regular or irregular.
 - Symptoms of: low cardiac output.
 - Precipitation of: angina & HF in susceptible patients.

SIGNS

- In Mobitz Type I: irregular pulse, Weak S₁.

¹ The Dutch doctor **Karel Frederik Wenckebach** (1864 - 1940), described the phenomenon.

- In Mobitz Type II:

1. PULSE:

- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **slow**, e.g. 30 – 50 / min. (according to AV block).

2. NECK VEINS:

- Multiple “a” waves: **double** or triple or quadruple the rate of pulse.

3. AUSCULTATION:

- Weak S₁.

ECG

- In Mobitz Type I:

- PR: Progressive prolongation until a QRS is dropped, **i.e.** until a p wave is not followed by a QRS complex.
Then, the PR interval returns to its normal duration and the sequence is repeated.

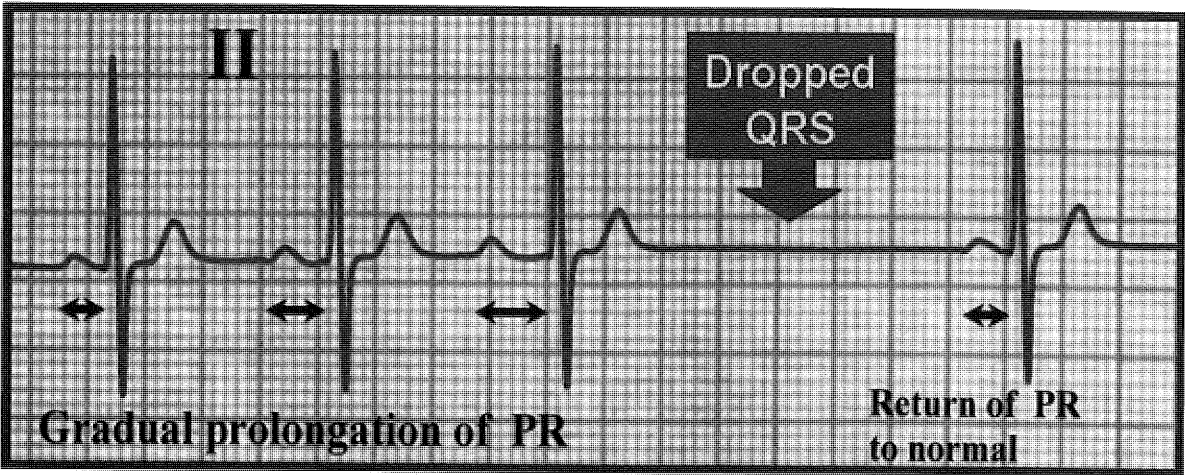
- In Mobitz Type II:

• QRS:

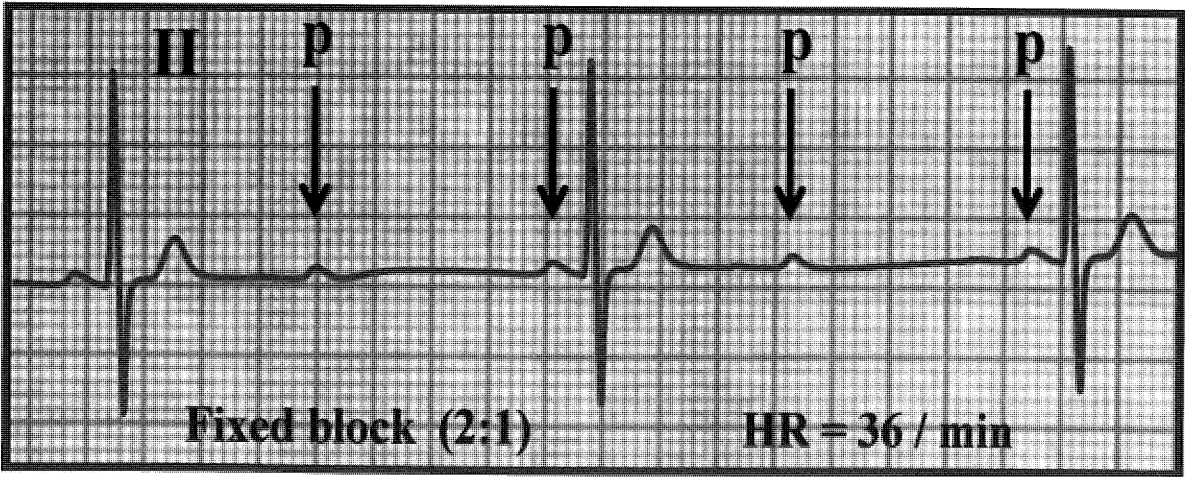
- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **slow**, e.g. 30 – 50 / min. (according to AV block).
- Duration: normal.

• P wave:

- Normal rate.
- 2, 3 or 4 p waves occur before each QRS (according to AV block).
- The block may be:
 - o Fixed (e.g. 2:1), **or**,
 - o Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).

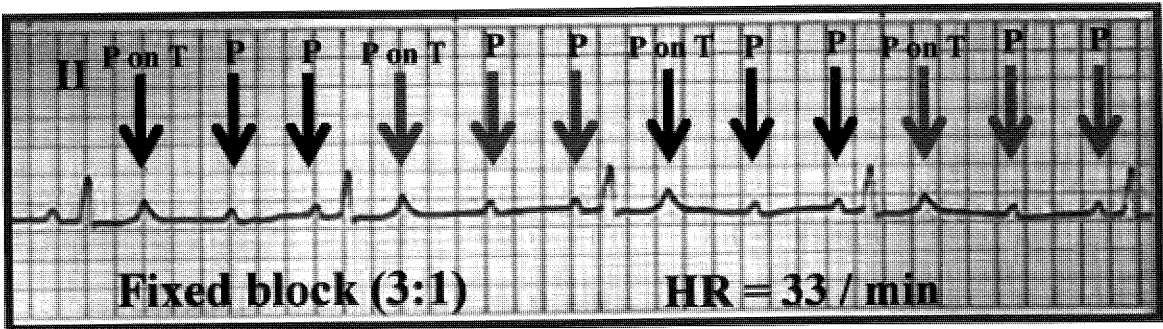
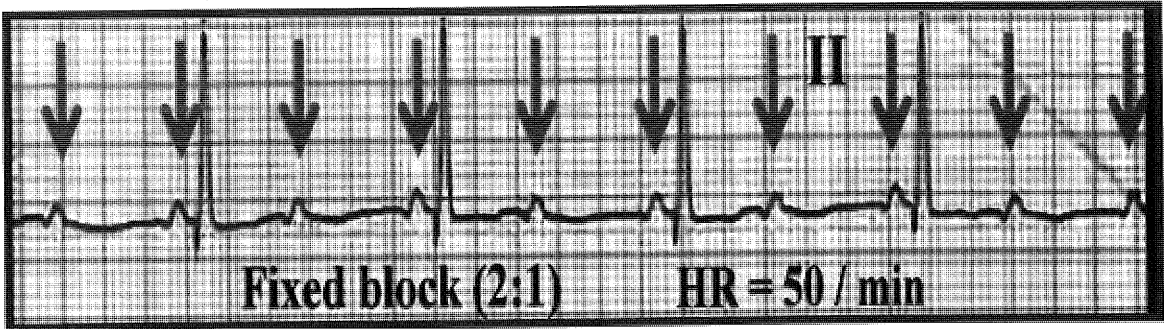


Mobitz Type I: (*Wenckebach phenomenon*)

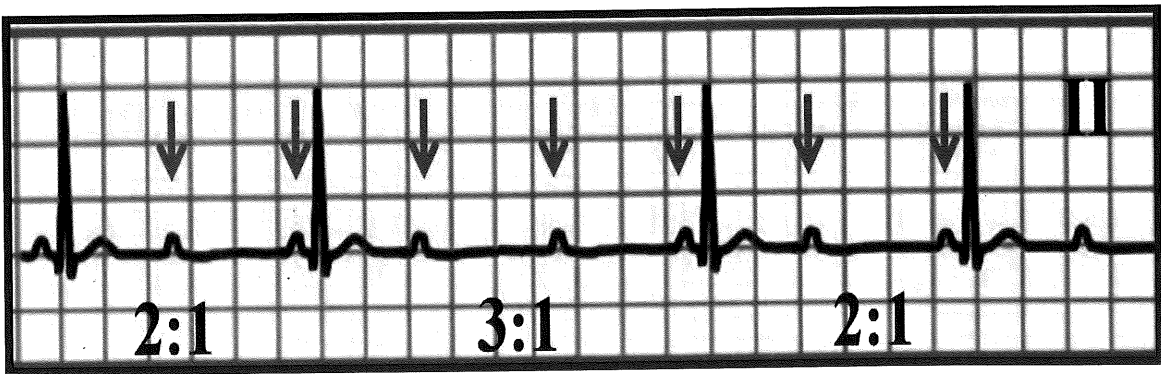


Mobitz Type II

Mobitz Type II (Fixed block)



Mobitz Type II (Variable block)



TREATMENT

1. TTT of: the cause.
2. Atropine: 1 mg IV.
3. PLUS: In Mobitz Type II, Artificial pacemaker.

Third degree (CHB)

DEFINITION

- No conduction of ALL impulses from the atria to the ventricles.
- How do the ventricles work ??
- They will be controlled by an: *Idioventricular pacemaker*.
- No relation between p waves and QRS complexes (*AV dissociation*).

CLINICAL PICTURE

SYMPTOMS

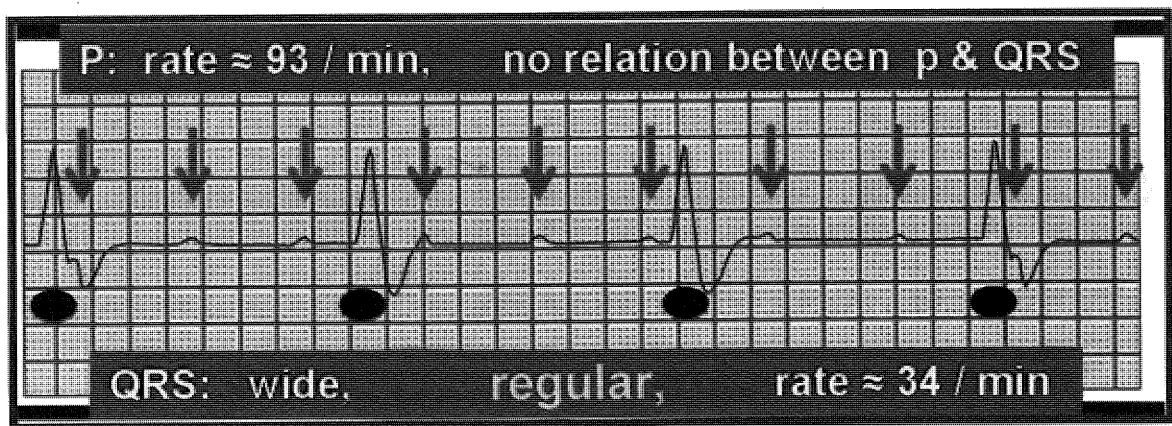
1. Symptoms of: *the cause*.
2. PALPITATION: regular.
3. Symptoms of: low cardiac output.
4. Precipitation of: angina & HF in susceptible patients.
5. ADAMS – STOKES ATTACKS:
 - They occur during transmission from one idioventricular focus to another.
 - There is: syncope, cyanosis, with *absent pulse, BP & heart sounds*.
 - If prolonged: convulsions, coma & death may occur.

SIGNS

1. PULSE:
 - Rhythm: **regular**.
 - Rate: **slow**, 30 – 40 / min.
2. NECK VEINS:
 - “a” waves: normal rate (60 – 100 / min) & more than the pulse rate.
 - OCCASIONAL CANNON WAVES.
3. AUSCULTATION:
 - OCCASIONAL CANNON SOUNDS.
 - Variable intensity of S₁.

ECG

- QRS:
 - Rhythm: regular.
 - Rate: 30 – 40 / min.
 - Duration: wide.
- P wave:
 - Normal in rate (60 – 100 / minute) & shape.
 - Comes before, after or is hidden by the QRS (AV dissociation).



TREATMENT

1. TTT of: *the cause.*
 2. Atropine: 1 mg IV.
 3. Artificial pacemaker.
- **Important question:** **"Self – assessment"**
 "What are the causes of sudden death ??"

SICK SINUS SYNDROME

- This is a clinically symptomatizing SAN dysfunction.

Etiology:

- Coronary heart disease
- Degenerative disease of the SAN.

Clinical Picture:

- Sinus bradycardia
- Sinus arrest , & atrial asystole.

Investigations:

- Ambulatory (Holter) ECG monitoring.

Treatment:

- Permanent pacemaker implantation.

Pre-excitation syndromes

- An accessory conduction pathway bypasses the AVN , leading to abnormal early activation of part of the ventricle . The remaining part of the ventricle will receive the normal impulse from the normal pathway.
- **Etiology:** congenital.
- **Clinical picture:**
 - Different arrhythmias: e.g. AF, flutter, or supraventricular tachycardia.
 - Sudden death.
- **ECG:**
 - Wolff-Parkinson-White syndrome (WPW):
"Wide QRS, Short PR interval, Delta wave".
- **Treatment:**
 - *Medical: control of the associated tachycardia.*
 - *Surgical: division of the accessory pathway.*

ANTI-ARRHYTHMIC DRUGS

Class	Mode of action	Subclass	Drug	Main uses	Side effects
Class I	Sodium channel blockers slowing the depolarization phase 0 depression	<u>IA</u> Moderate phase 0 depression	<u>Quinidine</u>	Broad spectrum	Idiosyncrasy Cinchonism N, V, D
			<u>Disopyramide</u>	Broad spectrum	Hypotension Anti-cholinergic effects
			<u>Procainamide</u>	Broad spectrum	Hypotension Lupus-like synd.
		<u>IB</u> Minimal phase 0 depression	<u>Lidocaine</u> Epanutin	Ventricular arrhythmias (AMI) (Digitalis toxicity) As lidocaine	Convulsions Confusion Skin rash BM depression Ataxia
		<u>IC</u> Marked phase 0 depression	<u>Propafenone</u>	Broad spectrum	<u>Pro-arrhythmia</u>
Class II	β -blockers		<u>Propranolol</u> & <u>Sotalol</u>	ST, SVT Atrial flutter AF Extrasystoles	Refer to the chapter of "angina "
Class III	Potassium channel Blockers slowing the repolarization (prolong the RF)		<u>Amiodarone</u>	Broad spectrum	Corneal deposits Thyroid problems Pulmonary fibrosis Hepatotoxicity
			<u>Bretylium</u>	Resistant VT	Hypotension
Class IV	<u>C</u> CBs		<u>Verapamil</u>	SVT Atrial flutter AF	Refer to the chapter of "angina "

Drugs with anti-arrhythmic action:

- 1. Adenosine: it ↓ conductivity in the AV node, so it is indicated in SVT, Atrial flutter.
- 2. Digitalis: it ↓ conductivity in the AV node, so it is indicated in SVT, Atrial flutter, AF.

CARDIOMYOPATHIES

DEFINITION

- Cardiomyopathy, which literally means "Heart muscle disease", is the deterioration of the function of the: MYOCARDIUM.
- A group of disorders that:
 - Primarily involve the: MYOCARDIUM.
 - Usually are: *subacute or chronic*.
 - **Are not the result of:**
congenital, valvular, pericardial, hypertensive or ischemic diseases.

CLASSIFICATION

1 Etiological Classification:

- a) Primary: Unknown cause (Idiopathic).
- b) Secondary: Due to a known cause or Associated with another disease.

2 Clinical Classification:

- a) Dilated (congestive) cardiomyopathy: *the most common*.
- b) Hypertrophic cardiomyopathy (HCM): *the second common*.
- c) Restrictive (non-dilated, non hypertrophic) cardiomyopathy.

DILATED CARDIOMYOPATHY

ETIOLOGY

a) Primary:

- Idiopathic.
- Familial.

b) Secondary:

1. Infective :
 - Viral: esp. Coxsackie B virus.
 - Others: Bacterial, protozoal.
2. Infiltration & granulomas:
 - Amyloidosis, Hemochromatosis, Sarcoidosis.
3. Immunologic: SLE, Scleroderma.
4. Iatrogenic & Toxic: Drugs (Doxorubicin), Toxins (Alcohol).
5. Endocrinal: DM, Acromegaly, Myxoedema.
6. Neurological:
 - Duchenne myopathy.
 - Friedreich's ataxia.
7. Peripartum cardiomyopathy.

- Important question: "Self – assessment"

"What is meant by: Ischemic cardiomyopathy" ??

PATHOPHYSIOLOGY

- Impaired Systolic function (↓ contractility).
- **Biventricular** dilatation.
 - In approximately 30% of patients with dilated cardiomyopathy:
There is an abnormality in the heart's electrical conducting system
(called an "intraventricular conduction delay" or bundle branch block).

CLINICAL PICTURE

- Manifestations of: BIVENTRICULAR FAILURE.
- Manifestations of: COMPLICATIONS:
 - *Embolic manifestations:* systemic & pulmonary.
 - *Arrhythmias,* e.g. AF, VT.
 - *Sudden death.*

INVESTIGATIONS

I. ECG:

- Biventricular enlargement.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- Arrhythmias, *e.g.* (*AF, VT*).
- Intraventricular conduction delay: BBB.

II. IMAGING:

1. CXR:

- Biventricular enlargement.
- Pulmonary congestion.

2. Echocardiography:

- SIZE: Biventricular dilatation.
- CONTRACTILITY: Impaired global contractile function.

3. Cardiac catheterization & angiocardiology:

- Biventricular dilatation.
- Impaired global contractile function.
- To exclude: CAD.

III. Other investigations for the cause:

- Endomyocardial biopsy: e.g. for Infiltration & granuloma.
- Serology: for SLE.
- Thyroid functions: for Myxoedema.

TREATMENT

1. Treatment of: HEART FAILURE e.g. ACEIs, β B, Diuretics, CRT.

2. Treatment of: COMPLICATIONS:

- Anti-coagulant drugs: *to prevent embolization.*
- Anti-arrhythmic drugs: *for arrhythmias.*
- Cardiac transplantation: *in refractory cases.*

3. Treatment of: CAUSE: e.g. Immunosuppressive drugs in SLE.

HYPERTROPHIC CARDIOMYOPATHY

ETIOLOGY

a) Primary:

- Idiopathic.
- Familial.

b) Secondary:

- Pheochromocytoma.

PATHOPHYSIOLOGY

- ASYMMETRIC LV HYPERTROPHY (SWT > FWT), leading to:
 - Impaired Diastolic function ($\downarrow$ compliance) $\rightarrow$ pulmonary congestion.
 - Obstruction of the LV outflow tract, causing low CO.
 - The septum may bulge into the RV cavity, causing giant a wave.

CLINICAL PICTURE

Symptoms

- Symptoms of: pulmonary congestion.
- Symptoms of: low CO.

Signs

General:

- Pulse: Jerky (sharply rising).
- Neck veins: giant a wave.

Cardiac:

A] Precordial examination:

- Signs of LV enlargement.

B] Auscultation:

- S₄: over the apex.
- Systolic ejection murmur: along the sternal border.
- Pansystolic murmur due to MR: over the apex.

COMPLICATIONS

1. Arrhythmias, *e.g.* AF, VT.
2. Sudden death: probably due to Ventricular arrhythmias.

INVESTIGATIONS

I. ECG:

- LV enlargement.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- Arrhythmias, *e.g.* (*AF, VT*).
- Abnormal Q waves: in inferior leads or lateral chest leads.

II. IMAGING:

1. CXR:

- LV enlargement.
- Pulmonary congestion.

2. Echocardiography:

- SIZE: Asymmetric LVH (SWT > FWT).
- CONTRACTILITY: ↑ contractile function of the LV.

3. Cardiac catheterization & angiocardiology:

- Usually not required: because non-invasive methods are diagnostic.
- ↑ LVDP: due to ↓ LV compliance.

TREATMENT

1. Treatment of: HEART FAILURE But: Avoid positive inotropics.
2. Treatment of: COMPLICATIONS:
 - *Anti-arrhythmic drugs*: *for arrhythmias.*
 - *ICD*: *to prevent sudden death.*
3. CCBs (Verapamil) or BB (Propranolol): to relax the septum.
4. SURGICAL TTT: resection of the hypertrophied septum.

RESTRICTIVE CARDIOMYOPATHY

ETIOLOGY

a) Primary:

- Endomyocardial fibrosis.
- Endomyocardial eosinophilic disease (Löffler's endocarditis).

b) Secondary:

1. Infiltration & granulomas:

- Amyloidosis, Hemochromatosis, Sarcoidosis.

2. Immunologic:

Scleroderma.

PATHOPHYSIOLOGY

- MYOCARDIAL FIBROSIS OR INFILTRATION, leading to:

- The **RV** is involved more than the LV.
- Impaired Diastolic function (↓ compliance) → Venous congestion.
- Mildly Impaired Systolic function (↓ contractility) → Low CO.
- The condition functionally resembles: Constrictive Pericarditis.

CLINICAL PICTURE

Symptoms

- Symptoms of: systemic congestion.
- Symptoms of: low CO.

Signs

General:

- GENERAL SIGNS OF SYSTEMIC CONGESTION.
- GENERAL SIGNS OF Low CO.

Cardiac:

A] Precordial examination:

- Signs of RV enlargement: late & mild.

B] Auscultation:

- S₄ over the tricuspid area & over the apex.

COMPLICATIONS

- Embolic manifestations: systemic & pulmonary.
- Arrhythmias, e.g. AF, VT.

INVESTIGATIONS

I. ECG:

- Low voltage.
- Diffuse nonspecific ST – T changes (Depressed ST, inverted or flat T).
- Arrhythmias, e.g. (AF, VT).

II. IMAGING:

1. CXR:

- RV enlargement: late & mild.

2. Echocardiography:

- SIZE: Symmetrical myocardial thickening.
- COMPLIANCE: Impaired global ventricular filling.

3. Cardiac catheterization & angiocardiology:

- Square root sign: “Early diastolic dip followed by a plateau” in: RV & LV ventricular pressure tracings.

III. Other investigations for the cause:

- Endomyocardial biopsy: e.g. for Infiltration, granuloma, fibrosis.

DIFFERENTIAL DIAGNOSIS

“Constrictive pericarditis”

	RCM	CP
PERICARDIAL KNOCK	Absent	May be present
PERICARDIAL CALCIFICATION	Absent	May be present
Cardiomegaly	Late & mild	Absent
CT & MRI	-----	Pericardial thickening
ENDOMYOCARDIAL BIOPSY	Diagnostic	-----
Exploratory thoracotomy	Diagnostic	Diagnostic

TREATMENT

1. Treatment of: HEART FAILURE e.g. ACEIs, Diuretics.
2. Treatment of: COMPLICATIONS:
 - Anti-coagulant drugs: to prevent embolization.
 - Anti-arrhythmic drugs: for arrhythmias.
 - Cardiac transplantation: in refractory cases.
3. Treatment of: CAUSE: e.g. Immunosuppressive drugs in Scleroderma.